BRAIN.

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Original Articles and Clinical Cases.

IMPERATIVE IDEAS.[1]

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I CANNOT do better than take for my text these words of a distinguished member of this Society:—"The slightest departures from the standard of mental health are to be studied, and not only the cases of patients who require to be kept in asylums. Indeed, the slightest cases are the more important in a scientific investigation."—(Hughlings Jackson.)

In speaking of imperative ideas I refer to those cases in which a person would not be regarded as insane, although the mental trouble may be as distressing as it is in actual insanity, and may at last become so pronounced as to render an asylum a relief to the patient himself. In our official classification of mental disorders we have not been in the habit of very clearly recognising, as a separate group, even this advanced stage, partly, no doubt, because if the patient becomes depressed, as frequently happens eventually, the case is classified under melancholia, or if he comes to interpret his imperative ideas as satanic or divine, his case is relegated

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to the head of "delusional insanity." In many instances such cases are diagnosed as *neurasthenia*, and there is no doubt that in some patients there is nervous exhaustion, but I think that the characteristic feature of the malady under which these persons suffer is too much lost sight of by so general and vague a term.

Symptoms.—With regard to the symptoms, they assume different forms. With some, the torment is that certain ideas or words arise with painful frequency and vividness. The thoughts which dominate the mind with morbid persistency are generally of an unusual and unwelcome character, and the words which the individual is strongly impelled to utter, are altogether contrary to his habit or desire, and if uttered, this is in spite of his strongly exercised will. Then there are persons who invariably touch some object in passing it in the course of an accustomed walk (*Délire du toucher*), the antithesis of which is seen in the dread of touching certain objects at all, a true mysophobia. Then there is a form of imperative ideas which Charcot and Magnan have termed onomatomania. These physicians have comprised under it a group of symptoms in which a word or a name plays the chief part, and have maintained that these mental troubles arise from what they call a loss of mental equilibrium. They also held that this condition is always associated with an hereditary taint of insanity, which I think is going too far. They included under this term, the agonising search for a particular name or word, a more or less irresistible impulse to repeat it again and again when found, or to attach a disastrous signification to certain words. (*Archives de Neurologie*, Sept., 1885, p. 137.)

One subdivision of imperative ideas, and a very striking one, is the insanity of doubt (*Maladie du doute*), and termed by the Germans Zweifelsucht and also Grübelsucht, or as we should say, beating one's brains about some question. Closely allied is the insanity of the metaphysicians—Schöpfungsfrage. According to the late Professor Ball, of Paris, the malady of the metaphysicians constitutes a subdivision of the malady of doubt. Then we have arithmomania, or the morbid desire to count without rhyme or reason, or to

make interminable calculations; to reckon up the number of books on the table, or the buttons on a waistcoat, or the same letters occurring in words. Almost endless, however, are the terms which have been introduced to label the particular form which imperative ideas assume. Certain alienists simply revel in coining terms for every imperative idea which they have ever met with. They give us not only agoraphobia and claustrophobia, acrophobia (the dread of being in high places), coprolalia (the involuntary use of obscene words), but astraphobia (the causeless fear of an approaching thunderstorm), zoöphobia (the fear of certain animals), oikophobia (the morbid fear of home), and to crown the complexity, bélonéphobia (the morbid fear of pins).

However allowable it may be for the sake of convenience thus to multiply terms, they must not distract our attention from the fundamental characteristics common to all imperative ideas. These are, their automatism, the overwhelming and recurring tendency to be haunted by a certain idea, to perform certain acts, or to use certain expressions against the will, *without delusion* (at least in the first instance), and indeed with a consciousness of the utter uselessness and absurdity of dwelling upon these ideas, and of the things which are done or said. Momentary relief may follow the discharge, but ultimately mental distress. The imperative conception may occur spontaneously, free from any external stimulus, and apparently (though sometimes only apparently) unconnected with antecedent thoughts.

It must also be remembered that although we speak of imperative ideas, some cases would be more correctly termed *imperative emotions*, and others *imperative impulses*. But that which I have more especially in view might be misunderstood if I used the latter terms, although the sufferer from the ideas in question suffers mental anguish in consequence, and often has irresistible impulses.

I am anxious to bring into prominent relief the absolute innocuousness and sanity of a large number of imperative ideas and acts. Take a few illustrations drawn from persons among my acquaintance, some of them being well-known physicians.

(1) A lady frequently wakes in the night with a sense of impending danger, conscious that there is no ground whatever for it. She obtains relief by giving three raps three times on the bedstead.

(2) A gentlemen, in attempting to open a door which he has no reason to suppose is stiff, for example in his own room, employs force to do so, and strikes the architrave in order to assist his efforts to ease the door. It has become a habit which he is unable to resist. I am not aware of any explanation.

(3) A medical friend has always an apprehension of something dreadful about to happen upon a door being closed, although not believing for a moment that anything will happen. Here again, no explanation can be found in any circumstance which previously happened calculated to associate the occurrence with an accident.

(4) Dr. ——— has the electric light in his house, and puts it out before retiring to bed. After going upstairs into his bedroom, he has troublesome doubts as to whether he has really done so. He returns in order to be quite certain, although he was certain when downstairs a few minutes before. Similar experiences are common, and may be explained by having performed an act so automatically that it had not been consciously registered; for, certainly, when a person deliberately and consciously performs an act, it could normally be recalled within a short period of time.

(5) Dr. ———, when on a height, experiences a strong and almost imperative tendency to throw himself over the edge. This does not arise from vertigo, nervousness or the slightest temptation to end his days. It is a blind impulse which he knows to be totally unreasonable, but which he finds it difficult to resist.

(6) Dr. ——— has, or had, an imperative idea, the exact opposite to Dr. Johnson's. He feels that he must avoid treading on the divisions between the flags of the pavement, or that something would happen of a serious nature. He is well aware of the absurdity of the idea.

The cases which I will now describe are, with some exceptions, illustrations of a graver mental condition, although the symptoms in the first instance, at least, are consistent with sanity, the subject of them being quite able to perceive their groundlessness and their ridiculous character.

(7) I had under observation a gentleman who was the last man in the world to use profane language, but who had the very greatest difficulty in preventing himself doing so in church and sometimes

while walking in the street without the slightest cause of irritation. His wife was surprised to see him give a sudden jerk from time to time, and was not aware of the cause. He, however, told me that these spasmodic movements arose from his attempt to rid himself of his trouble and avoid using bad language, and as a matter of fact the incipient motor act was checked at that stage. For this painful condition one has for long been accustomed to employ more especially the term *obsession*, as indeed, the French do for all imperative ideas. There is, however, the objection to the word, in relation to a patient, that it encourages the idea, only too common, that some demoniacal agency is the cause of the trouble. In fact, in the case now spoken of, the gentleman was disposed to regard his trouble in this light, and was partially relieved when informed that it was nothing of the kind. I will only add that he was ultimately greatly benefited by a sharp attack of gout.

(8) Next, as regards the tendency to touch objects, there is the well-known instance of Dr. Johnson, whose mental peculiarity is thus described by Boswell :—"At that time the broad flagging at each side of the streets was not universally adopted (in London) and stone posts were in fashion to prevent the annoyance of carriages. Upon every post as he passed along he deliberately laid his hand; but missing one of them when he had got to some distance, he seemed suddenly to recollect himself, and immediately returning back, carefully performed the accustomed ceremony, and resumed his former course, not omitting one, till he gained the crossing" ("Life," vol. i., p. 497). Dr. Johnson also illustrated the counting mania, and indeed I am not sure that the touching of the posts in the streets was not also due to this arithmetical passion. We are told of his anxious care to go out or in at a door or passage, by a certain number of steps from a certain point, or at least so that his left foot should constantly make the first actual movement when he came close to the door or passage. Boswell adds: — "Upon innumerable occasions I have observed him suddenly stop, and then seem to count his steps with a deep earnestness; and when he had neglected, or gone wrong, in this sort of magical movement, I have seen him go back again, put himself in a proper posture to begin the ceremony, and having gone through it, break from his abstraction, walk briskly on, and join his companion."

Johnson, it should be added, laboured under an hereditary taint of insanity, and had time permitted I could have given an instructive account of his mental history.

(9) In the following case, that of a lady, sent to me by Dr. Strangman Grubb, of Ealing, the most prominent and distressing symptom was the incessant tendency to count a certain number of times before performing the simplest act. It can readily be imagined what mental fatigue this imperious necessity occasioned. To be haunted by what the late Professor Ball called an "intellectual impulse" would be bad enough for a few days, but to be the sport of this tyrannical suggestion for years (in fact she had been six when I was consulted about her), could be no other than an intolerable burden. She described to me how even in the night, she could not turn over in bed (unless she did it with great rapidity), nor remove the watch from under her pillow, without counting. In the morning she generally experienced the greatest difficulty in getting out of bed without being obliged to go through the same numerical process. Her friends would hear her step on the staircase on coming down to breakfast, and then several small raps were made by her feet, whilst she was prevented proceeding further in consequence of having to count up to a certain number before descending another step. She says that if anyone had been behind her and said "Come along, you go downstairs," she would have been able to do so, without having to pass through this tedious process. Imagine her now at the breakfast table, wishing to take up the teapot; a considerable time would elapse before she was able to seize the handle, the only means of accomplishing this feat being the same wearisome process of counting, generally to ten or multiples of ten. Suppose now she took a walk, she could not open the front door without counting, and having passed into the street she would probably be impelled to return simply to touch the door handle. She had no pleasure in looking at the shop windows or at pictures, for she had to count a certain number of times before her enemy allowed her to regard them. She was frequently compelled to count the number of times she breathed, and she could not avoid counting her steps as she walked along the road. Again, in saying her prayers, she had not proceeded far, before she was under the dire necessity of returning to the first word and going through the prayer again. At church her difficulties were great, as may be supposed, and to take the prayer book into her hand was an exhausting operation from the act of counting which it involved. Sometimes she would put the book down again, and take it up in a different way. To open a drawer was a very serious matter indeed. Before blowing out a candle she had to count so many times, and when using her needle, she put it into

her work as often as twenty times before making one stitch. The malady of doubt entered largely into this state of mind. When reading, she often worried over the doubt as to the paragraph in the page (or if there were two columns, which of them) she should read first.

When asked whether she had ever thought there was poison in her food she said not, but she answered rather doubtfully. On the following day I received a letter from her to say, that although she had never thought that her food had been purposely poisoned, she had sometimes a nervous fear that something *might* have accidentally got into it, adding that she had sometimes refrained from eating things from just the same kind of feeling which had on many occasions prevented her saying the things she wished to say. She remarked in regard to both the feeling of hesitation and the habit of counting, that "when very bad it produces a feeling of mental tension, which is very trying, not to say painful, and which makes it extremely difficult to occupy myself with any ease or pleasure. One feeling which has made me afraid of breaking through this habit of counting has been a fear lest the thought—sometimes one and sometimes another—which has connected itself with the counting might fix itself in my mind and be worse than the counting."

About nine years ago, when in church, something was wrong with the heating apparatus, hot air escaped, and she felt as if she would be suffocated if she remained. Everything became dark and indistinct to her as she went out. In consequence of this she was depressed for a week, and some of her subsequent troubles followed upon it. The dread from which she had never previously suffered, of being buried alive arose; and she was afraid of walking in her sleep into some churchyard, and being supposed to be dead, when only in a state of somnambulism. She pictured to herself all the horrors of a conscious interment.

Such is a rapid description of the trial under which this unfortunate lady laboured for many years. Perfectly conscious of this strange mental condition, and very bright and intelligent, she was able to discourse upon it, and to suggest what she thought was the process of thought by which she came to preface almost every act of her life by counting. She compared it to a boy running a race, but unable to start until someone calls out "One, two, three and away!" She regards this as the immediate origin of her habit, and that she did in the first instance, and does perhaps even now count, with this object.

Although there is no insane inheritance known, it is of interest

to note that one of her brothers died an epileptic, and one of her sisters, who is living, has epileptic fits. A law student, I shall shortly mention, had a family history of epilepsy as well as insanity. In a remarkable article in the *Annales Médico-psychologiques* (Jan. 1890), by Dr. Culerre, the association of onomatomania with epilepsy is insisted upon. In the case I have brought before you there have been no fits of any kind, and she has never fainted, although turning deathly pale when hearing the account of the death of Mr. Bishop, the thought-reader, connected as it was with the suspicion that the autopsy was made when he was not really dead. She does not appear to have had attacks of *petit-mal*, but I elicited the fact that she has at times the feeling of "I wonder am I really doing this?" She has never suffered from vertigo. She has experienced the not uncommon sensation of having been under precisely the same circumstances and in the same place on former occasions. She was a nervous child, but she assures me that she was not naturally undecided, and although not making up her mind very quickly, she had no tendency to vacillation. There has been no hallucination of any of the senses and no delusion. I have recently heard that she has recovered.

The next case is an example of mysophobia and the malady of doubt.

(10) This patient (Watts) came under Dr. de Watteville's care at the West End Hospital for Nervous Diseases, in February, 1893. His attention was not drawn to any mental trouble beyond general nervousness and a feeling of being equal to his work. He regarded the case as one of *neurasthenia*.

Some months afterwards, Dr. de Watteville asked me to see this patient. His mental symptoms were these: He said that his great trouble was that on going to do a thing he always had a sense of doing it wrongly, and after posting a letter or putting something into a drawer, he had an invincible misgiving as to whether it was correctly done. He felt that there was no rational ground for this misgiving. He was also greatly worried as to whether he had taken anything away in his hand from any place which he should not have taken. After getting out of an omnibus he began to think that he had left something behind him; he said that he knew perfectly well that he had not, and yet he could not help having the idea presented to and tormenting his mind. Again he felt from time to time, he knew not why, that he must look up to the ceiling of the room he happened to

be in. He had fancies about his clothes being dirty, and often looked at his hands to see whether they were quite clean. He did not complain of mental depression.

On the *physical* side there was slight tremor of the hands. The pupils were dilated, sluggish and equal. His knee jerks were very brisk. With the exception of dreaming his sleep was good. His habits were temperate and he was not a smoker.

Family History.—His mother suffered from "nervousness," and he considered that he inherited it from her. He was the youngest of twelve children, four of whom died of phthisis, and a brother committed suicide. He was not aware of any insane or epileptic ancestor.

Some months later he became worse, and it was quite a relief to him to be allowed to come into Bethlem Hospital as a voluntary boarder.

I will now describe a very striking case illustrating imperative ideas not amounting to delusion in the first instance, but ultimately becoming so.

(11) A. F., aged 58, sent to me by Dr. Wakefield, was able, when I first examined him, to laugh at the notions which arose in his mind, and he would say that of course he knew there was nothing in them. He said that two years previously, the idea came into his mind when he took the Eucharist that blood would come into his mouth. This idea had entirely left him, but only to give place to others. He was a Roman Catholic, and when young he was engaged as an acolite about the altar, and he had, while in the church, strongly impressed upon his mind the pictures and images of the crucifixion. To this he himself attributed a feeling of swelling and pain in the palms of his hands, and his attention was in consequence frequently directed to them. In the night if he awoke he was under the impression that he himself was Christ, but when he got up in the morning he shook off the idea, and was distressed to think that he should have entertained it even for a moment. No stigmata ever made their appearance, but it is not difficult to believe that they might have done so in the course of time, for he got to suspect that blood was flowing from his hands. This led his sister to put out of sight a picture which he had bought representing the crucifixion.

But the most absurd, and to him the most mortifying imperative idea of all, was that he was pregnant. It was not when he first mentioned it to me a delusion, for he could not help laughing when he told me. It originated partly in a dream and partly

from reading in a newspaper that a young woman had said that if *she* were *enceinte*, she should drown herself.

Some sensory disturbance was mixed up with his idea, for he said it seemed to him as if his abdomen was of greater size, and yet he added, "I know it isn't." Sometimes he had to resist the idea that flashed into his mind, that he was a woman, and felt that he should like to shut himself up in his room and not see a soul, for very shame. Above all things he was afraid when walking in the street that he should meet a pregnant woman. Well, after a while, he really believed that he was pregnant; he no longer smiled at the idea, but pulled a very long face indeed.

With regard to his *family history*, his mother was subject to mental depression, but not in an asylum. He has a brother and two sisters, and they are mentally healthy. There does not appear to have been any epilepsy in the family.

To complete his history, I should add, that after some time he refused his food, averred there was some obstruction of the bowels, and it became necessary to place him in a private asylum, where he died of pneumonia in about ten days.

(12) *Case of Miss* ——.—This is the case of a young lady who has laboured for some time under the dread of touching anything for fear of infecting others who might touch the same article, and I fear I can hardly maintain that her ideas do not now amount to delusions. The first symptoms arose after influenza. There is at the present time in her mind the fear of infecting herself as well as others, although she does not say that she *knows* it would be so. The result is that the act of washing her hands in the morning is repeated, immediately, again and again, and although she does not own to counting, her friends think that this idea enters into her course of ablutions. She invariably passes the sponge over the upper surface of the arm, one side, then the under surface, and lastly the other side of the limb. She forces herself to lie awake, lest in her sleep she should put forth her hand and therefore contaminate some object near her. What seems a strange inconsistency, is that she is obliged at times to touch objects, feeling in a vague way that if she did not, something dreadful would happen. She has on one occasion burnt her clothes, fearing that they have been infected by her. She says she would have washed them, but knew she would not be allowed to do so.

Then she suffers from the malady of doubt. Endless questions arise in her mind. Which gloves shall I put on? The brown ones? The black ones? and so on. She will spend

twenty minutes at her drawer in this state of indecision, conscious that it is foolish to do so. I may here remark that the hopeless indecision under which some sane people labour is akin to, if it does not fall under this form of imperative ideas.

As a fact, this young lady scrubs her hands and arms so fiercely that they are quite rough and discoloured afterwards. She keeps one part of the towel for the face, another for the hand, another for the wrist, and so forth. When I advised her not to think about it, she replied: "But then what would happen, if unconsciously I *did* infect the towel?"

It is important to note that she herself states that this morbid dread began in consequence of having warts on the hands, and hence dreading she would infect other people with them.

While admitting that this case more nearly approaches insanity than sanity, I should not dream of signing a certificate for her.

(13) *Case of Miss B.*—The next case is one of mysophobia of a very similar character to the last. Her imperative ideas have certainly crossed the rubicon between those which are not credited and those which are. I only mention it as illustrating the later stage, although she is not in an asylum. It is interesting as combining the dread of contagion with the onomatomania of Charcot and Magnan. It is important also as showing that a careful analysis of imperative ideas and acts will often explain their origin. Thus while reading a book or newspaper she will suddenly evince signs of irritation and disgust, and it becomes necessary to relieve her of the annoyance by removing the book. Now it is found that a particular word has been the source of the excitement. Nothing in the word itself could explain the effect which it produces. A complete knowledge of her history, however, unravels the mystery. The explanation is this: a compound word happens to contain the syllable which is the name of an individual to whom many years ago she took a strong aversion, although she had little acquaintance with him. She would never enter the room where he was. It is long since he died, but she still dislikes to see or hear any name that contains his, even although in part. This aversion has ramified so completely into her life that it is sometimes very difficult to trace a particular act to it, but there is reason to think that it is always there. It may be traced in the idea of dangerous contamination with certain objects. She objects to people whom she meets touching her, and this on careful sifting turns out to be part of the same irresistible dislike to the individual in question. Hence the

demand she makes to have certain things washed. When she comes in from a walk she invariably washes her hands and face. She is most particular in washing away dust. Frantic washing of hands and arms is sometimes preceded by maledictory expressions. Occasionally words come into her mind in this wise: she will startle a friend by saying "verdigris," and then she will say in an appealing manner: "Thinking of it won't make verdigris come, will it?" She will also ask questions again and again which bear on her fear of contamination, saying, "Let me say it again; did so and so touch my dress?"

We see then in this case an illustration of the fear of contamination with objects, in themselves absolutely clean, but associated by some accidental circumstance with an individual or a long past event in the patient's life.

As to her family history, there is no proof of insanity or epilepsy on either her father's or mother's side; both families have been healthy and long lived. A cousin, however, is almost an idiot. An uncle was paralysed. The patient herself had a very severe illness in her infancy, but her childhood had passed when the unreasonable aversion to the gentleman had developed. In character she is impulsive, affectionate, generous in thought, but selfish in action, and with considerable religious feeling and sense of duty, not always carried out into practice. She is exceedingly truthful and unsuspicious. Intellectually she is regarded in society as perfectly sane, exceptional in wit, fond of science and music, and possessed of an excellent memory, with however, a great lack of patient application. There are no hallucinations of any of the senses.

I now pass on to describe the symptoms of a very striking example of imperative ideas assuming the form of Charcot and Magnan's onomatomania, and the malady of doubt in general.

(14) A boy of 19 (sent to me by Dr. Barlow), a law student who had gained distinction at school, and matriculated at the London University, met in the course of his reading with the expression, "*It was not incompatible.*" Shortly afterwards he came across the German words, "*Ich liebe es nicht.*" Now, it struck him that the negative in the former sentence preceded, and in the latter succeeded the most important word. He then began to puzzle himself about negatives in general. Whatever he read raised the question in his mind as to the construction of sentences in which a negative occurred. It became an all-

important, an absorbing problem. It interferred with his reading and working. The burden of his life was to place the negative in its right order, whatever that might be. One day he said to his father, "If it wasn't for that confounded negative I could master Blackstone and any other law book." At one time he was puzzling himself over the question, "Why haven't *we* cold blood, like some animals?"

The great danger is that he will become irresolute and vacillating in his actions, the continuance of the original mental difficulty weakening his will power and threatening to paralyse practical life altogether by the number of questions and doubts which arise in his mind as to the possible course to take in a particular instance. The proverbial three courses will not suffice long for those who become the sport of innumerable doubts and endless questionings. The law student's career becomes itself "a negative," in the presence of his morbid fascination for negatives.

With regard to heredity, I find that his father, a man of fine physique, is subject to attacks of mental depression, while the young man's maternal uncle has been for many years an epileptic.

Indeed, we cannot fail to be struck with the frequent occurrence of epilepsy in the families of persons labouring under imperative ideas. Napoleon, himself an epileptic, could not help counting the number of windows in the houses he passed. At certain points these cases seem to be closely allied to epilepsy. Now, it would be felt quite inappropriate to apply the term insanity to such a case as that of the law student. We must, indeed, admit that we can hardly regard the individual who cannot control his thoughts in the way that he wishes to control and direct them as a person of perfectly sound mind; but no "last will and testament" would be set aside in this case, or on the ground that a testator had an invincible desire to touch certain objects, or an intense repugnance to touch certain articles of furniture, or that he had an intolerable fascination for a particular word, or that certain ideas were associated with a special dress with which no one else had the same feeling. Nor would such morbid habits of thought and association of ideas go far to sustain the plea of insanity and irresponsibility in a criminal trial.

I now come to the last section of my paper:—

Cerebral Conditions Accompanying Imperative Ideas.

If now we consider what are the correlative changes in the cortex of the brain, we must say at once that no microscopical examination would disclose any pathological lesion associated with the morbid mental condition I have described. We are assisted, however, by starting with Laycock's doctrine of the reflex or automatic function of the cerebral cortex, as enunciated by him in a paper written fifty years ago. In this very original paper, read at York in 1844, before the British Association, he says of certain morbid mental conditions, that "their detection is sometimes difficult, because the patient being keenly conscious of his infirmities will conceal them. If, however, by what is termed the association of ideas, the morbid action of vesicular neurine be brought within the current of his thoughts, he becomes utterly powerless to resist it, as much so as the electro-biologised (hypnotised) to resist the suggestions presented to their minds. The formation of these *substrata* is due to the fixity of the mind on one idea or class of ideas, at a time when, from morbid changes induced in the vesicular neurine (as by undue mental labour, intense emotional excitement, want of repose, the development of a dormant predisposition, and the like), it is unusually susceptible to the operation of the unconsciously constructing mind, so that the fixed ideas become deeply writ, as it were, on the vesicular neurine, in the same way as acquired instincts, habits, &c.; and are, in fact, as difficult to remove.

Laycock writes: "The principle of reflex cerebral action will be found to be of the utmost importance to the elucidation of the relation of thought and will to the encephalon and to the comprehension of the marked changes they and it undergo in all forms of insanity."

In explaining some instances of imperative ideas by accidental association, it will very often be found that this association has arisen from their contiguity under some powerful emotional impression. I know a physician who cannot think of his father's funeral, which occurred some forty years ago,

without the troublesome mental image of a white terrier being, to his annoyance, called up, simply because one appeared during the ceremony when the coffin was about to be lowered to the grave. Clearly this is due to the vividness with which the incongruous circumstance was at the moment registered upon his cortical tabula, never to be erased.

Laycock taught that "there is not only cerebral evolution in regard to healthy brain development, but an equally general inverse law of *dis*-volution (*sic*), equally important in studying mental disorders."

Laycock's theory fits in with the latent influence of association, not only of ideas, but of certain impressions received through the special senses, for in some forms of imperative ideas these become accidentally and sequentially connected in an extraordinary manner. The individual wishes to destroy this newly formed and accidental association, but he becomes absolutely dominated by it. Laycock, in his now little read article, insists that "the true explanation of the association of ideas is to be found in the doctrine of the reflex functions of the brain" (*B. & F. Med. Review*, January, 1845, p. 311), and yet, strange to say, no one except Dr. Jackson has done him justice, many have not once referred to him, and some have not heard of him!

Laycock recognised the control exercised by the cerebral cortex over sensory impressions, but does not appear to have anticipated current doctrines in regard to the control of one portion of the cortex over another, or positive and negative conditions of the mental functions. These doctrines it remained for Anstie, Monro, and above all Hughlings Jackson (who always expresses his indebtedness to them, and to Herbert Spencer), to teach.

I believe that Dr. Jackson has not in any of his writings referred specifically to imperative ideas other than as insane delusions, which according to him "signify evolution going on in the remains of a mutilated nervous system, as certainly as the patient's belief, when sane, signified evolution going on in the entire (*i.e.*) unmutilated nervous system." (Leeds Address by Dr. Jackson, 1889.) There is in short a "dis-volution" of the highest cerebral centres, but as Dr. Jackson

points out "there are all gradations traceable as a consequence of disease beginning in the highest centres, from such slight depths of dissolution as those which permit nearly normal actions, to dissolutions so deep as to afford no evidence of mentation." (*Med. Press and Circular*, December 9, 1871.)

Accepting this hypothesis, in addition to Laycock's great fundamental doctrine of the reflex or automatic function of the whole cortex, we seem to find an answer to the question I began with, what are the cerebral conditions accompanying imperative ideas? The most automatic is no longer under the control of the voluntary or least automatic cerebral levels or layers, or in Jacksonese, "reversals of evolution have occurred, being reductions from the least to the most organized of the highest cerebral centres."

Take the simplest and most frequent illustration of imperative ideas. Some lines of poetry come unbidden into the mind. There is nothing remarkable in that. They recur half-a-dozen times in the course of the morning. Nor is there anything very remarkable in that. If, however, they recur again and again during the rest of the day, the limit of healthy cerebral action may have been passed, and the individual may discover two things, *first*, that when a word in one of the lines presents itself, he has no peace or relief until they are all mentally (and perhaps verbally) completed, and *secondly*, that to his annoyance the relief is only short-lived, and the same obligation as before soon arises and tyrannises over him. What at first was a pleasure has become a nuisance. Here then is the earliest stage of an imperative act of memory. In the majority of cases a good dinner, a good night, and exercise in the open air will break the spell by reinforcing inhibitory power. But in others the automatic process returns and is as inevitable in its action as the going off of an alarum. Worst of all, words now present themselves, and recur again and again, which not only harass by their persistent frequency, but by their unsavoury character. They do not, or may not, find vocal expression at first, but sooner or later a motor discharge takes place which is irresistible.

With regard to those cases in which there is a morbid *dread of dirt*, I do not know that we can say more, in many instances, than that there is an exaggeration of the scrupulous cleanliness which in a marked degree characterises some persons who are in perfect health, but the origin of the imperative idea may occasionally be traced to some affection of the skin which has necessitated the attention being drawn to it, and has at last induced a morbid state of mind, not of introspection, but of "extero-spection." Should the general mental power be lowered, or a hereditary predisposition exist, this tendency to attend to the state of the skin becomes a passion, and results in the needless and wearisome washings of the body or the dress. Indeed, it too often happens that at last the patient no longer perceives the folly of these ablutions, but labours under an actual delusion.

With regard to the singular habit of *touching* certain objects without apparent reason, it is sometimes explained (by persons labouring under it) as due to a fear lest something will happen if they do not do it; but in other cases as an irrepressible habit without any motive whatever, often performed almost unconsciously as well as automatically. There may, however, be a conscious struggle between the voluntary or least automatic, and the most automatic cerebral action, the former being too feeble to control the latter. It often happens that ideas of touching are associated with ideas of doubt. In fact "doubting Thomas" touches in order to satisfy his doubts.

Among the illustrations I have given of imperative ideas, the most curious, perhaps, is that of automatic counting. And yet the act of counting is one so peculiarly automatic that it is natural it should be performed by persons with weakened higher centres. We have seen how in the case of a lady, she likened her state to that of the boy who is assisted in running a race by hearing or uttering numbers before making the start.

Of the malady of doubt I have said that a troublesome, although slight degree of it, is not uncommon in perfectly sane people. It was only the other day that an officer of

an asylum engaged in laboratory work, told me that not unfrequently after shutting the door the last thing at night and having no doubt that it was shut, he would return once or perhaps twice to satisfy himself that this was the case. In the same way I have known persons open an envelope in which they had placed a cheque and had exercised great care that the date and signature were correct, in order to satisfy themselves that they were so. And again, a man may give to a friend, verbally, the number of a particular street quite correctly, and have no hesitation about it, but within five minutes may have misgivings, visualise another number, and begin to fear that he has said 20 when he ought to have said 40. Is this because the registration of the number has been feeble? This seems hardly likely, because one has noticed these doubts arise in the prime of life, when the brain would receive impressions clearly and retain them, as well as in persons more advanced in age.

I once knew a gentleman, tall and massive in form, who developed an imperative idea, the origin of which was clear enough. He began by thinking that if he died when from home, this might happen in a house in which it would be impossible for the undertaker to get his coffin down the stairs. The idea after a time haunted him and became very troublesome, and moreover, he said, that at last the worry was not confined to himself, and that he never met a man of tall stature without perplexing himself with the question whether his staircase would allow of his coffin being taken down stairs. I may add that a son and a grandson committed suicide. It cannot be said that Bacon's axiom, "He that questioneth much shall learn much," receives confirmation from these cases, for in a man's questioning thus, his last state is worse than the first.

What strikes one forcibly in regard to most, if not all, imperative ideas is that between them and ordinary ideas the difference is one of degree, and that it is a most difficult thing to determine when the boundary line has been passed. This on the psychological side. And on the physical side, what we want above all things to know is what cerebral change, degeneration, or dissolution has occurred by which

the man who to-day is conscious of and smiles at an imperative idea, gravely believes it to-morrow, and labours under a systematised delusion.

Granting that there are sub-levels and superimposed levels in the highest centres themselves, we must, I suppose, assume that in the case of imperative conceptions without actual insanity, no pathological changes have induced dissolution of the very highest level or area, and that this only happens later on, when the patient with more extended dissolution no longer distinguishes between true and false ideas. The predominating idea has, in fact, become part of his own being, and not one extraneous to himself upon which he can look down as distinct from his personality—his ego.

In short and finally, is the explanation to be found in the degree or extent of dissolution in the same range or level of the highest centres, or is it to be found in different depths of dissolution in these centres ?[1]

[1] Those who are familiar with German and French writers, will find that their recognition of the mental phenomena described in this article has been distinctly clearer than our own.

ON THE ASSOCIATION OF DISEASE OF THE SPINAL CORD WITH PERNICIOUS ANÆMIA.

BY H. M. BOWMAN, M.D.

ATTENTION has recently been directed by certain German observers to the fact that in patients suffering from severe progressive anæmia, of the type usually known as "pernicious," symptoms now and then develop pointing to an affection of the spinal cord; and it has been shown that these symptoms, though varying somewhat in some respects, are in others fairly constant, and are associated with changes in the cord of such striking similarity as to justify the cases being regarded as belonging to the same pathological group.

This association of severe anæmia with lesions of the spinal cord was first pointed out in 1887 by Lichtheim, who described three instances which he had observed, and he laid stress upon the special character of the changes found in the cord as distinguishing these cases from tabes, to which there was some general clinical resemblance. These cases were subsequently published in detail by Minnich,[1] together with three others which he had since observed. In these six cases, which were characterised by the usual symptoms of pernicious anæmia, the nervous phenomena bore a distant resemblance to tabes in some instances, in others rather suggested disseminated sclerosis, the most constantly observed being a progressive muscular weakness, which was found in every case, and subjective disturbances of sensation in the extremities which were twice wanting. Ataxy was noted in half the number, but was associated with loss of the knee-jerk in one case only, which was also unique in the fact that the pupil had lost its reaction to light. The gait, in most cases, could not be accurately

[1] *Zeitschrift für Klin. Medicin*, xxi., 1892.

noted owing to the marked motor weakness. Objective sensory disturbances, varying between partial anæsthesia to all forms of stimuli and slight disturbance of sensibility to one or more forms only, were observed in five cases, and were most marked in the legs. The condition of the knee-jerk varied considerably, it being reported to be absent twice, diminished once, normal once, and exaggerated twice. Involuntary muscular spasms occurred twice. Loss of control over the sphincters was present toward the end in half the cases. All these cases were males at about middle life, and in all the fatal termination occurred within a year of the first development of the nervous symptoms. The degenerations found in the spinal cord showed some difference in regard to extent, but there was close agreement in the distribution. In every case there was degeneration in the posterior columns, most marked in Goll's tract, but also involving more or less of the postero-external columns, but in the latter case leaving a strip of undegenerated white matter on the inner surface of the posterior gray cornua. The posterior roots and Lissauer's tract were invariably intact. In four instances the lateral columns were also affected, the degeneration being situated chiefly in the outer and posterior portion, and in three of these cases similar changes were also observed in the anterior columns, chiefly in the situation of the direct pyramidal tract. It was pointed out that in spite of the marked degeneration in the posterior columns there was never found to be any shrinking such as occurs in tabes, which may probably be explained by the nature of the lesion, which rather suggested a subacute process than a chronic sclerosis.

Von Noorden[1] published another instance in a woman of 59, who was suffering from progressive anæmia, and in the course of this disease developed spinal symptoms, indicated by weakness and paræsthesiæ of all the extremities, impaired sensibility to touch in the legs, loss of the knee-jerk, and towards the end disturbances of the sphincters. The pupil reactions were normal, and ataxy was not obvious. The spinal cord showed "total degeneration" of the posterior

[1] *Charité Annalen*, 1891.

columns, not involving the posterior roots, and degeneration in the crossed pyramidal tracts.

In a case described by Eisenlohr[1] as an example of pernicous anæmia probably due to atrophic changes in the gastro-intestinal tract, were noticed motor weakness and paræsthesiæ in the extremities, a spastic condition of the legs, loss of knee-jerk without obvious ataxy or affection of the pupil, and, later, loss of control over the sphincters. He found degeneration in the posterior columns of the cord, closely resembling that usually occurring in tabes, as well as scattered foci in the lateral columns. His patient was a man aged 59.

Two interesting cases have recently been recorded by Nonne[2]; the first being a man aged 48, who, while suffering from pernicious anæmia, developed nervous symptoms characterised by numbness and weakness in the lower extremities, definite ataxy in all limbs, occasional girdle sensation and spasms in the legs, normal pupil reactions, slight transient analgesia in the legs and, later, loss of control over the sphincters. The knee-jerk varied; at first it was diminished, then was entirely lost, and later was again distinctly obtained.

His second case, a man aged 57, had suffered from uncertainty in walking, occasional girdle feeling, and numbness in the hands and feet for some months *before* anæmia was noted. At first he had ataxic gait, slight anæsthesia and analgesia of the legs and fingers, which ultimately cleared up, and greatly diminished knee-jerks. Later, anæmia had become very prominent, the knee-jerks could be readily obtained, and motor weakness had increased, ataxy being no longer demonstrable, and there was incontinence of urine. The light reflex of the pupil was never lost.

In both these instances the changes found in the cord were most marked in the posterior columns, especially in Goll's tract, which presented the appearances usually found in ascending degenerations in the upper dorsal and cervical regions. The postero-external columns showed a variable degree of change, but everywhere the zone bordering the

[1] *Deut. Medicin. Wochenschrift*, 1892.
[2] *Archiv für Psychiatrie*, xxv., 1893.

inner aspect of the posterior cornua, the posterior nerve roots, and Lissauer's tract were unaffected. The lateral columns were extensively involved in the region of the direct cerebellar and crossed pyramidal tracts, and there was slight degeneration in the direct pyramidal tracts. In the anterior, and partly in the lateral, columns the process was found to consist in the more or less complete coalescence of small dotted foci of degeneration, apparently of comparatively recent formation.

The case which I will now describe was recently in the National Hospital, under the care of Dr. Hughlings Jackson, to whose kindness I am indebted for this opportunity of publishing it. In the main points of this case, both clinical and pathological, will be found a close resemblance to the ten cases alluded to above, especially, perhaps, to the two described by Nonne, and it must clearly be included in the same group.

Elizabeth R., aged 53, married, three children, was admitted on July 12, 1892. Sallowness was first noticed two years previously, during which time she had also suffered from headache, and recently she had become much paler. Nine months ago the legs began to feel very weak, and for one month a feeling of "pins and needles" was constantly present in the legs.

Her previous health had been good. There was nothing pointing to syphilis beyond a single miscarriage. Menopause a year ago, but severe "flooding" two months before admission.

On admission the mucous membranes and skin were very anæmic, the latter having a lemon yellow tint. General nutrition fair, there being abundant, though not excessive, subcutaneous fat. She was unable to raise her head from the pillow without giddiness. Appetite fair, bowels acting regularly, tongue clean. Urine non-albuminous. The lungs presented no evidence of disease. The heart was not dilated, and no hæmic bruit was audible. Liver dulness commenced at the sixth rib, and the edge was felt more than an inch below the costal arch. The spleen just descended below the ribs on deep inspiration.

Nervous System.—All voluntary movements of the trunk and extremities could be performed, though somewhat feebly, no actual paralysis being demonstrable. From patients being un-

able to stand the gait could not be observed, but there was no ataxy or tremor in the movements of the limbs.

Sensibility was affected as follows :—There was blunting to very light tactile and painful stimuli in the finger tips, and slight tactile anæsthesia in the legs below the knees, analgesia being less evident and confined to the toes.

The knee-jerks were decidedly brisk, wrist-jerks were readily obtained, but no ankle clonus could be elicited. The plantar and abdominal reflexes were brisk.

No affection of the cranial nerves was found.

The eyes were free from nystagmus ; the pupil reactions normal; distant vision good, though accommodation was weak ; retinal hæmorrhages of various sizes were found in the fundus, as well as some white patches of effusion.

Examination of the blood showed the red cells to be 19·5 per cent., and hæmoglobin 20 per cent. of normal ; micro- and poikilocytes were found, but no large nucleated red cells, or "eosinophil" cells were discovered (these were never found at any subsequent time) ; white cells were not increased.

The mental condition was unaffected. Control over the sphincters was complete. No girdle feeling or pains in the limbs were complained of.

Treatment by iron having been tried for a time during which the anæmia gradually became worse, arsenic was substituted with apparently striking results, as will be seen below.

August 22.—Slight improvement in general condition, anæmia being less intense. Blood estimation showed an increase of red cells to 32 per cent. of normal. Since admission the temperature had remained about normal, only occasionally reaching 100·4, the maximum observed. Less motor weakness. Sensation practically unaltered, but it was noted that there was distinct impairment of position sense in the toes, most noticeably at the big toe joints. Localisation of stimuli was correct. Subjective sensations of "pins and needles" still complained of in the hands and legs. Reflexes unaltered.

September 21.—Marked improvement in general condition, anæmia being much less intense, and patient was able to sit up out of bed, being able to walk a short distance with the help of a nurse, or in a "go-cart." No ataxy was observed when she stood up or walked. Sensibility was greatly improved, there being no impairment to tactile or painful stimuli, though a touch had a "numb" feeling on the feet and legs. The blood value had increased to 58 per cent. of normal.

Fig. 1.

Fig. 5.

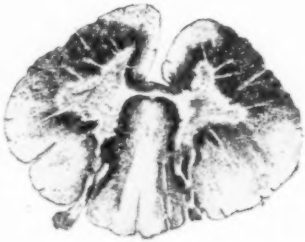

Fig. 2.

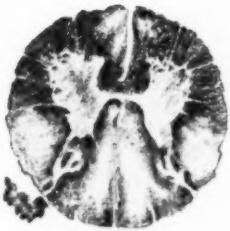

Fig. 6.

Fig. 3.

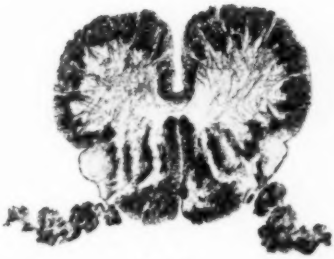

Fig. 7.

Fig. 4.

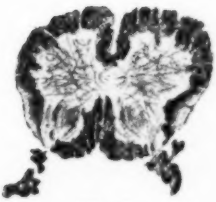

Fig. 8.

October 7.—Still further improvement. The blood reached about 82 per cent. of its normal value in red cells. No hæmorrhages were found in the fundus oculi.

October 29.—Patient was now able to walk without support, her gait, beyond a certain amount of feebleness, showing no special alteration from health. Sensation normal. Spleen not palpable now. Complexion was rather sallow, though anæmia was not noticeable. Blood estimation gave proportion of red cells at 87 per cent.

Patient was soon after this sent out to a convalescent home, and then was kept under treatment as an out-patient. Early in 1893, however, she was found to be worse again, and on March 3 she was re-admitted, having been unable to walk for a month, and giving a history of occasional retention of urine. At the time of re-admission her general condition was very much the same as was noted when first she came under treatment, there being marked anæmia, with a sallow tint of skin, but no evidence of disease of the chief thoracic and abdominal viscera. The spleen could not be felt.

Examination of the nervous system gave the following results :—She was quite unable to stand, being only just able to raise her body into a sitting posture by great effort. Movements of the arms were rather feeble; those of the legs markedly so, there being just power of raising the heel from the bed. On executing fine movements with closed eyes slight ataxy was found; no tremor present. The legs were subject to sudden and painful involuntary spasms.

Sensation was considerably disturbed, there being impaired sensibility to touch, pain, and temperature in the lower limbs, most definite towards the feet, and gradually fading towards the trunk, which was only slightly affected. Localisation was very incorrect, and position sense was completely lost at the toe, ankle and knee joints.

Reflexes were in the same condition as was observed in the first note.

The sphincters were unaffected. The *blood* showed red cells in the proportion of 68 per cent.

July 23.—Much weaker and more anæmic. Blood estimation gave 37 per cent. of red cells. She could not turn over in bed unassisted, arm movements were feeble, leg movements markedly so. Ataxy was now very definite in the arms. The legs were semiflexed and rigid, passive extension causing great pain, and spasmodic jerkings were more painful and frequent. The impair-

ment of sensibility was more definite, and was found on the fingers for the first time. Knee-jerks very brisk; a non-persistent slow clonus obtained at the ankle; superficial reflexes exaggerated. Pupil reactions normal. No retinal hæmorrhages.

August 11.—Was much worse. Vomiting was troublesome, and interfered with medicinal treatment. The temperature was constantly above normal, frequently reaching 101°. Retention, followed by incontinence of urine, and later of fæces, was observed. There was œdema of the feet. Blood value about 25 per cent. of normal.

September 5.—Patient was now quite helpless, being unable even to put her arms out of bed without help. There was wandering of the mind at night. Incontinence was complete. Œdema of the legs extended to the knees. Anæmia was very marked, red cells being in proportion of 18 per cent.

September 13.—Extreme anæmia—red cells being reduced to 12 per cent. Patient constantly in condition of muttering delirium. Temperature constantly subnormal. Both eyes showed numerous retinal hæmorrhages. Death occurred on the 16th.

At the autopsy there was great anæmia of all viscera. The pleuræ contained free fluid, and the bases of the lungs were congested. The heart was pale, but not obviously fatty. The liver large, pale, and fairly firm—no free iron reaction was obtained either on fresh section or in stained specimens. The spleen was not enlarged. There was a cyst communicating with the duodenum, apparently of inflamatory origin, situated in the head of the pancreas. In the fresh cord a gray discolouration was seen on section, occupying the posterior columns, and most marked in the upper dorsal region. Otherwise the cord and brain appeared normal, except for extreme pallor.

After hardening in Müller's fluid a very extensive degenerative process was apparent in the cord, involving not only the posterior columns, but also more or less of the lateral and anterior columns. Sections were stained by the Pal-Weigert method, and in carmine, and under a low power there was found to be the following distribution of the lesions :—

(1) *Upper Sacral.*—The degeneration is here limited to a small patch at the outer and posterior margin of the lateral columns, which is more extensive and more complete on the right side, though on neither side is the process far advanced (see fig 8).[1]

[1] In this, and in all the other figures, the degeneration has been made to appear slightly more intense than is actually the case.

(2) *Mid-lumbar.*—In the lateral column of either side is a patch of degeneration occupying the posterior portion of the column, and presenting a roughly triangular outline, with the base, where the change is most complete, extending to the periphery of the cord. On all other aspects the degenerated area is surrounded by healthy white matter, except at the extreme posterior extremity.

In the posterior column the only apparent change is in the form of an irregular collection of foci at the base of the right tract of Goll (see fig. 7).

(3) *Upper lumbar.*—In each lateral column is a patch of degeneration having a similar distribution to the above section, except in being of larger extent, and in the densest degeneration being in the centre of the area. Except at the outer side there is healthy white matter on every aspect into which the degenerated portion of the column gradually merges.

In the posterior columns changes are found occupying the greater portion of the tracts of Goll, the most complete change being in the centre of each area at the margins of which it becomes patchy in distribution and fades gradually into healthy tissue. The base of the tract is unaffected. In the postero-external columns the changes are slight, and chiefly confined to the basal part, being most marked on the left side.

Small foci of degeneration occur in the direct pyramidal tracts especially in the left (see fig. 6).

(4) *Lower dorsal.*—The lateral columns are here very extensively involved, the healthy tissue, into which the degeneration very gradually fades, being considerably the smaller part of the columns.

The greater part of the posterior columns is also involved, the healthy white matter being limited to a narrow strip bordering the inner aspect of the posterior cornua and the gray commissure, and a wedge-shaped area at the base of the median fissure. Scattered islets of partly degenerated white matter are found in the affected tissue, though for the most part the change is very complete.

In the anterior columns degeneration is found in the direct pyramidal tracts, where the change is most advanced, but it also extends outwards along the margin of the column for some distance, gradually fading and assuming a dotted distribution (see fig. 5).

(5) *Mid-dorsal.*—In most respects the changes here are similar to those in the last section, though they are more complete and more uniform, and there are fewer healthy fibres at the base of Goll's tract.

There is a small island of degeneration in the left antero-lateral tract (see fig. 4).

(6) *Upper Jorsal.*—The changes in the lateral columns, though still involving a large area, are now rather less extensive, and less complete, except toward the periphery of the cord. In general the distribution is similar to that described above.

In the posterior columns the degeneration closely resembles a secondary ascending process in distribution, being almost confined to Goll's tract. The changes extend into the inner aspect of the postero-external columns, which are also affected slightly at their bases.

The anterior columns are chiefly affected at their median and anterior margins. The antero-lateral tracts are free (see fig. 3).

(7) *Lower cervical.*—In the lateral columns the greater part of the white matter is still involved, though the affected area is dotted here and there with islets of comparatively healthy fibres, and is for the greater part less completely changed. The distribution is principally marginal.

The posterior columns show very dense changes, pretty sharply mapping out the limits of Goll's tract, which, except at the base and near the commissure is almost completely degenerated. The postero-external columns present slight changes along their inner aspect, and more advanced changes near their bases, especially on the left side.

The anterior columns are affected to the same extent as in the last section (see fig. 2).

(8) *Mid-cervical.*—There is less degeneration in the lateral columns, the peripheral region being the part most affected, and the changes are generally much less advanced.

In the posterior columns Goll's tract is almost entirely affected, and the changes are very advanced. The outer columns are more extensively involved at their bases than in the last section, though here, as in every other instance, the degenerated areas are separated from the posterior gray cornua by a strip of healthy white matter. On the right side there is also a collection of foci of degeneration at the anterior part of the column.

The anterior columns are affected much as in the last, though the change is almost limited to their median aspect (see fig. 1).

(9) *At the lower part of the decussation* there is a wedge-shaped area of degeneration occupying the posterior column, chiefly in the postero-median portion. In each lateral column is a small peripheral area which has undergone a similar change. The pyramidal fibres are affected in a somewhat irregular manner, and degenerated fibres can be detected in the decussation.

(10) *Just above the decussation* the changes are found to have a similar distribution, but are less extensive, and less complete. In the pyramidal tracts the degeneration has a dotted distribution.

In no part of the cord can any shrinking of the affected columns be detected.

Under a higher power of the microscope the degenerative process is found to vary considerably in the different regions. Taking the periphery of an affected area in the anterior or lateral columns, where the change is least dense, and may fairly be assumed to be in an early stage, the most obvious alteration found in the nerve fibres themselves. At first a few isolated fibres may be seen among apparently healthy structures, the medullary sheaths of which are swollen, often to three or four times the diameter of a healthy fibre, and which take Weigert's stain less readily, so as to appear paler than usual; the axis cylinder still appearing to be normal. Among such fibres are found several empty spaces where the affected fibres have dropped out, probably in the cutting and staining processes. The interstitial tissue in such situations does not show any very obvious change.

On examining a part where the degeneration is apparently more advanced it is found that the proportion of healthy fibres is much smaller, the majority presenting some degree of the changes mentioned above, extreme degrees of swelling of the medullary sheaths being shown, and from the relative diminution in the number of fibres a larger proportion have probably been destroyed or have dropped out of the section. The interstitial tissue is somewhat thickened, and a number of nuclei are seen scattered about between the nervous elements.

In the parts of the section where the most intense changes have taken place, it is found that the interstitial tissue is largely in excess, such nerve fibres as are present being isolated and presenting more or less swelling of their myelin, while a considerable number of nuclei can be seen here and there. In the upper part of the cord a still greater increase of connective tissue elements is found in Goll's tract, where nerve fibres are in places entirely wanting, and here the interstitial tissue has a denser appearance than elsewhere.

The blood vessels are found to be affected in proportion to the degree of interstitial change, being apparently healthy in areas where the nerve elements are solely or chiefly involved, and presenting very obvious changes in the more densely degenerated

parts. The earliest alteration consists in a proliferation of nuclei in the sheath of the smaller arterioles, while in a later stage the sheath is crammed with cells, the lumen of the vessel remaining normal. In the most advanced areas of degeneration (in Goll's tract) the walls of the arterioles are greatly thickened and the lumen much reduced, in some instances being occluded.

The posterior roots are everywhere intact. The posterior cornua are also free from change, though the degeneration in the lateral columns extends up to them at some levels. They are always bordered by a strip of healthy white matter on their inner surface.

The ganglion cells, the gray matter generally, and the pia mater are entirely free from change. The peripheral nerves, unfortunately, could not be examined.

From the above description it will be seen that the degenerative changes are for the most part not strictly limited to definite tracts of long fibres, also that appearances which may reasonably be regarded as indicating an early stage of the process are met with at every level of the cord, from the mid-lumbar to the highest cervical region. From these facts it may be inferred that the process is a primary one, affecting all three columns, though at least the denser degeneration found in the situation of the crossed pyramidal tracts at the lower levels, and in Goll's tract above the mid-dorsal level are probably of the nature of secondary descending and ascending lesions respectively.

The minute changes suggest that the process is rather of a parenchymatous than an interstitial character, at least primarily.

I have to acknowledge my indebtedness to Dr. W. S. Colman for much kind help and advice in the pathological investigation of the cord.

Remarks.—Clinically it will be seen this case presents many points of resemblance to the group of cases previously referred to, especially in the gradual onset of nervous symptoms commencing with subjective sensory disturbances and progressive motor weakness, and in the comparatively short course from the first onset of these symptoms to the fatal termination; though in this one respect my case was somewhat unusual in its longer duration, which extended over a year and nine months, including, however, a period of three months, during which there was an almost complete freedom from symptoms.

In the later stages of these eleven cases the nervous phenomena presented considerable variations, a few cases (those of Lichtheim) bearing some resemblance to tabes, others rather approaching the type of disseminated sclerosis, and to this latter group my case belongs. The resemblance to disseminated is not at all close, as such important symptoms as volitional tremor, nystagmus, and articulatory changes were absent. A careful study of this group of cases does not reveal any one symptom or complex of symptoms which would justify their being regarded as forming a distinct clinical group, beyond the one fact that there was present, in addition to the nervous phenomena, an anæmia of severe and progressive type, and they must, I think, be included in the large and rather ill-defined class of "combined postero-lateral scleroses," the differences in the symptoms depending upon the relative extent of the lesion in the posterior and lateral columns respectively.

In his article on "Ataxic Paraplegia," Gowers[1] states that the disease is usually characterised, among other things, by a gradual onset, a slow course without special tendency to shorten life, and by an intact condition of sensibility even in the late stages, as well by an excessive activity of the deep reflexes. He adds, however, that in rare cases there may be sensory impairment, and in some the knee-jerk is lost. In these anæmic cases, on the other hand, the course was always rapid, being usually less than a year, sensory disturbance was observed in every instance, and the knee-jerk presented many variations, being absent throughout in four cases, and temporarily in two others, and in no instance was it present in great excess. Too much importance must not be attached to these differences, since many cases of postero-lateral sclerosis have now been recorded in which anæsthesiæ or loss of knee-jerk, or both, have been present. Probably the presence of the grave blood disease is quite sufficient to account for the rapid course of the anæmic cases, though a similar short duration of the disease has been more than once observed where no special allusion is made to

[1] "Diseases of the Nervous System," 2nd edit., vol. i, p 453.

the presence of anæmia (Strümpell,[1] Ormerod,[2] and others.) On these grounds, this group of anæmic cases may be taken to belong to the class of postero-lateral scleroses, so far as their clinical features are concerned.

In considering the pathology of these cases two questions arise: (1) Are the lesions found to be constant? As regards the *extent* of the degeneration this is certainly not the case, as in two instances the change was found in the posterior columns only, in two others in the posterior and lateral columns, while in the remaining seven cases all three white columns of the cord were involved, and even in these the extent of degeneration varied. In every instance, however, the posterior columns were most extensively affected, so that this apparent difference may depend upon the duration and severity of the process. With regard to the distribution of the changes; if we neglect Eisenlohr's case in which the distribution was reported to be very similar to that usually found in tabes, and Von Noorden's, where there was "total degeneration" of the posterior columns, though leaving the posterior nerve roots intact, in all the remaining cases certain features are found to be constantly present. Thus the degeneration in the posterior columns is most advanced and most extensive in the tract of Goll, which, in the lower levels, is frequently affected alone, while the changes in the postero-lateral-columns are less complete, usually less extensive, and are most marked in the middle or upper levels of the cord. In every case, and throughout the whole length of the cords, a zone of healthy white matter is found bordering the inner aspect of the posterior cornua. The lesion in the lateral columns, while chiefly affecting the crossed pyramidal and direct cerebellar tracts is not limited to any definite systems of long fibres. The microscopical appearances appear to be similar in most respects.

(2) Are these changes to be regarded as specific? This must be answered in the negative, since the distribution of the lesions is precisely similar to that found in numbers of cases of combined sclerosis, unassociated with anæmia, and

[1] *Archiv für Psychiatrie*, 1881.
[2] *St. Bart. Hosp. Reports*, xxix., 1894.

closely resembles that described by Gowers as typical of ataxic paraplegia. The microscopic changes bear many points of resemblance to the lesion of subacute myelitis, only in the most densely affected parts presenting any notable degree of sclerosis, and here the change is probably in great part of a secondary nature. Precisely similar changes have been observed in cases of postero-lateral sclerosis without anæmia, as in Ormerod's case alluded to above.

From the above considerations it will be seen that the pathological changes found in these cases cannot be regarded as in any way characteristic, which was also observed of the clinical course and symptoms.

It remains to be considered what is the relation between the anæmia and the spinal lesions. At first sight it seems tempting to assume that the degenerations in the cord are secondary to the grave blood state, and if this could be shown to be the case it would be a matter of some importance, as possibly throwing some light on the difficult question of the ætiology of some forms of spinal degeneration at least. Unfortunately this is not easy to prove. Minnich, in summing up his six cases, was disposed to doubt the dependence of the spinal disease upon the anæmia, since the latter was often of variable degree and sometimes of short duration, at the time of onset of spinal symptoms. He suggested that both trains of symptoms were dependent upon some unknown toxic influence. The same observer made an examination of twelve cords from patients who had died from pernicious anæmia without any nervous symptoms having been observed, and in five of them some changes were discovered, in some cases bearing a general resemblance to the lesions found in his six published cases. This is suggestive of there being some very close relation between the blood state and the spinal disease.

One argument against the anæmia being primary is to be found in the fact that in Nonne's second case the symptoms of spinal disease distinctly preceded the onset of anæmia.

On the other hand many points may be raised in favour of this view (that the spinal disease is the result, at least in some instances, of the blood state):—

First, it is seen that with one exception the anæmia dated back beyond the first observed spinal symptoms. In my case some degree of anæmia had been present for fifteen months before nervous symptoms occurred.

Secondly, it is worthy of remark that in my case during the gradual recovery of the patient under treatment the nervous symptoms also abated, until at the time of her discharge, when the condition of the blood was little below the normal standard, there was nothing that could be demonstrated beyond a slightly feeble gait and an exaggerated condition of the knee-jerk; but that, with the relapse of the anæmia, all the previous nervous phenomena returned and gradually increased with it up to the time of death.

Lastly, there is considerable evidence that in the course of anæmia of severe degree, no matter of what type, certain disturbances of the nervous system do now and then occur. On this question Gowers[1] alludes to the well-known fact that optic neuritis can occasionally be found in chlorosis, as an instance of the influence of general anæmia upon the nervous centres, and he mentions weakness of the legs, sometimes amounting to complete paralysis as another rare symptom. In anæmia from loss of blood, also, he describes paraplegia, usually affecting the motor functions only, as being sometimes observed. Through the courtesy of Dr. Buzzard I am enabled to refer to a case (as yet unpublished) which recently came under his care at the National Hospital.

The patient, a young girl, was the subject of extreme chlorosis, and when first observed there was found to be complete absence of the knee-jerks, even on employment of the "reinforcement method." During the course of treatment by iron, however, the jerks gradually returned, and they could be obtained without difficulty when convalescence was completed.

When it is considered that the blood changes and the general signs and symptoms in pernicious anæmia are usually much more grave than is commonly the case in chlorosis, or simple anæmia, it is only reasonable to expect that

[1] *Loc. cit.*

nervous phenomena, which are occasionally found in the latter conditions, should now and then be met with in the course of the former, and also that when they do occur they would be of a more severe type.

The eleven cases were all of the type of pernicious anæmia, though probably the cause of this condition was different in a large proportion; thus two cases were ascribed to bothriocephali, three or four to gastro-intestinal disturbances, including one case where atrophy of the glands of the gastro-intestinal mucous membrane was found, and one case followed a metrorrhagia. In the remainder no probable cause was discovered.

In conclusion, I would suggest that these cases, in which spinal symptoms of a somewhat anomalous type are associated with grave and progressive anæmia, be regarded as belonging clinically and pathologically to the group of postero-lateral scleroses (ataxic paraplegia) and that until further light be thrown upon the subject it would be unwise to be too positive as to the relation of the nervous symptoms to the altered blood state, though I am inclined to regard the anæmia as primary, and probably the cause of the changes in the cord.

PATHOLOGY AND TREATMENT OF GRAVES' DISEASE.

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GENTLEMEN,—The investigations and discussions with regard to the pathology and treatment of Graves' disease have reached a point that makes it possible to present several important issues of scientific and practical interest in a moderately definite form.

The essential questions are these: Are the symptoms of Graves' disease mainly toxic in origin, and due to an excessive or perverted secretion from a diseased thyroid gland; or does the affection belong to the class of the so-called neuroses; or, finally, are the symptoms due to an irritation of the thyroidal and cervical nerves?

If it can be determined that the thyroid is directly or indirectly the principal cause of the disease, then a strong case is made out for the removal of that gland by surgical means, provided no strong contra-indications are present. As a matter of fact, this operation has already been done in a large number of cases, and on the whole with encouraging results. The most successful results are, however, as we too well know, liable to be published first, and the proper position of thyroidectomy in the treatment of Graves' disease has yet to be established. My own experience with two cases has made me feel the need of caution in recommending this treatment.

The writer who first called attention to the importance of the thyroidal theory, and who has given it perhaps the strongest support, is Mœbius.[1] Other writers of promi-

[1] Schmidt's *Jahrb.*, vol. ccx., July 15, 1886; *Zeitschr. für Nervenheilk.*, 1891; and other papers.

nence have skilfully maintained the same view, two of the latest of them being Dr. Greenfield, of London, who makes this topic the subject of the Bradshaw lecture of 1893,[1] and Rehu, of Frankfort,[2] who has had much practical experience with the surgical treatment of these cases.

The argument for the thyroidal-secretion theory, drawn from the successful results of operations, is not as convincing as it at first sight seems. It happens often enough that morbid processes are kept up by influences which were not their real cause, and cease when these influences are withdrawn. Furthermore, the symptoms of Graves' disease have sometimes been favourably influenced by such operations as the draining of a cyst of the thyroid (Rehu), or removing a sarcoma (Tillaux) without doing much if anything to the secreting portions of the gland; and operations for the removal of irritations from the nasal cavity have also been remarkably effective in a few cases in giving the first impulse toward cure.

The thyroidal theory has taken, in general, one of two forms: (*a*) That the amount of the thyroid secretion is increased, and, being poured into the blood, acts as a poison to the nervous system, and perhaps to other tissues; (*b*) That the thyroid secretion, whether increased or not in quantity, is altered in character (v. Mœbius). The latter view was proposed to meet the objection that some of the symptoms of Graves' disease may appear before any recognisable glandular enlargement has occurred. The idea of an altered secretion is ingenious and suggestive, but is not yet substantiated. A great deal of careful chemical and physiological work must be done before we can decide such a question. Greenfield says that the secretion of the diseased gland is liable to become mucinoid, instead of colloid, in character, but this does not greatly help us. Now that Graves' disease is more commonly treated by thyroidectomy than formerly, it should be possible to obtain fresh material for direct experimentation, but until this has

[1] *Lancet*, December 9, 1893.
[2] *Deutsche med. Wochenschrift*, 1894, p. 265.

been done we ought to admit that we know simply nothing of the action of perverted thyroid secretion. It is difficult to believe that just the same perversion would occur in each case, always giving rise to much the same symptoms, *i.e.*, the symptoms of the typical Graves' disease, and it would not be proper to accept the arguments adduced in favour of the increased secretion theory as necessarily supporting the perverted secretion theory. It has also been suggested as possible (Maude, *Lancet*, 1893), that even when no obvious enlargement of the thyroid has occurred the secretion may be increased in quantity, but before much weight is attached to this view, as yet unverified, the validity of the thyroid theory in general deserves further scrutiny.

(1) It is pointed out that Graves' disease presents various points of similarity or antithesis to the myxœdematous cachexia which follows the removal of the thyroid. Thus, in the first stages of this cachexia, symptoms of nervous excitability make their appearance, which are not unlike some of those characteristic of exophthalmic goitre. The temperature may be elevated, tremors are often present, and muscular jerkings sometimes occurring in broken series.

(2) Besides these points of resemblance, there are also points of contrast which are very marked at first sight—though, perhaps, they are in reality rather superficial—between the symptoms of Graves' disease and those of the later stages of cachexia thyreopriva, or the ordinary condition of a myxœdematous patient. This contrast is tersely expressed by Mœbius,[1] who says: "In the one case we have enlargement, in the other diminution in the size of the thyroid gland; in the one a rapid, in the other a slow heart beat; in the one we have a fine skin, warmer than usual and inclined to sweat, while in the other the skin is thick, cold, and dry: on the one hand we see increased mental irritability, a condition of irritable weakness, on the other slowness and dulness of the mind."

It is certainly obvious that in these two conditions we have symptoms which are in a measure the antithesis of each

[1] *Zeitschr. für Nervenheilkunde*, 1891.

other, but this fact should not be admitted as indicating more than it really does indicate. Many of the strictly nervous symptoms of Graves' disease, such as the tremor, irritability, increased heart beat, and flushing of the skin, are met with in conditions of nervous excitement where no question of increase in the thyroid secretion comes in at all. As regards the differences in the condition of the skin, it is doubtful if there is any real contrast between the state induced by a relaxed vascular contraction and increased activity of the sweat glands, and that due to such manifest structural changes as are seen in myxœdema, where the diminution in perspiration is, for all we know, a secondary element. Fortunately, we have in the extracts of animal thyroids, now so largely in use for myxœdema and other states, a means of studying the action of the thyroid carefully and at leisure.

To some extent this has already been done, and the results seem, in some, though not in all respects, confirmatory of the thyroidal theory of the origin of Graves' disease. These facts have been passed in review by Murray (*Lancet*, London, 1893), Greenfield, and others. An excess of thyroid extract seems to cause, quite regularly, even in healthy persons, to judge from the few experiments reported, an increase in the pulse-rate and temperature, and often diuresis with increased secretion of urea. Myxœdematous patients are sometimes very susceptible to this substance, and sometimes suffer from serious prostration, with anginoid and neuralgic symptoms, as the result of a dose which other patients bear with ease.[1] In such cases, however, important changes in the metabolism partly related to the previous myxœdematous state, are rapidly going on, attended with great loss of weight, and it may be that these changes, or possibly the absorption of toxic products, are in part the cause of the symptoms noted. Loss of weight is, indeed, apt to occur even with healthy persons who are taking thyroid preparations, and yet it is noteworthy that patients with Graves' disease are not especially prone to emaciation as one might expect them to be if poisoned with

[1] See, for example, Shattuck: *Boston Medical and Surgical Journal*, 1894.

thyroid secretion. It is obvious that the absorption of too much secretion or of an altered secretion might cause some of the symptoms of Graves' disease, but it cannot be said that anything like a clear case is made out in favour of the whole complex being due to that cause.

It has been said in this connection that thyroid extracts given to patients with Graves' disease seem as a rule rather to increase than to diminish the symptoms already present, but I do not consider this argument to be of much value. Inasmuch as the thyroid preparations increase the heart-beat in normal persons, it is obvious they would be likely to increase the tachycardia of Graves' disease, but even this is not a regular occurrence. I have been giving of late five to ten grains daily of desiccated thyroid to a patient with Graves' disease without any increase in her palpitation or nervous symptoms, and, in fact, with relief to some of them, and others have had the same experience.

A striking case of this kind has been published by Owen.[1] This physician attempted to treat a case of typical Graves' disease of twenty years' standing by the use of thyroid extracts. The patient's wife, by mistake, administered a quarter of a pound of chopped thyroid on each of two successive days. This was followed by nausea and eructations, vertigo, insomnia, swelling of the face and legs, but nothing of greater import. A week later the treatment was resumed, and a quarter of a lobe of sheep's gland was given daily, as originally ordered. Steady improvement soon began, which progressed so far that the pulse fell from 126 to 76 beats per minute, the exophthalmos almost disappeared, and the patient became able to do heavy work. A single fact like this is of great value, and seems to show that the improvement in the general nutrition which has now been shown to occasionally follow the use of the thyroid in non-myxœdematous cases may sometimes be made to serve a good turn in the treatment of Graves'[2] disease.

[1] *British Medical Journal*, Dec. 2, 1893.

[2] See a paper by the writer in the *Boston Medical and Surgical Journal*, February 15, 1894.

It occurred to me last winter while watching a case of thyroidectomy for Graves' disease,[1] that some of the ill effects of this operation may be due to the pouring out of a large quantity of thyroid secretion from the cut surface of the gland into the fresh wound of the neck. In this case a glairy substance actually exuded into the dressing. If the reports may be trusted these ill effects do not, to be sure, always occur, but in four cases (one of them a case of ordinary goitre) which have come under my observation, the patients were very ill, and the pulse very rapid. One of the patients died at the end of a week, though doing well in all respects except for restlessness, nausea, and cardiac weakness.

(3) Another argument adduced by Greenfield in favour of the thyroidal theory is that the histological examination of the thyroid shows that in Graves' disease we have evidence of an increase of the gland with signs of considerable activity. The thyroid of the adult is made up mainly of small and irregular spaces, lined with epithelium and containing colloid. In Graves' disease the structure becomes arborescent, suggesting that of a secreting gland, the epithelium becomes higher, and the colloid disappears or is changed into a substance of different character.

There is, however, another and quite different significance which may, with quite equal reason, be attached to these changes. It was pointed out several years ago by Halsted and Welch, of John Hopkins' Hospital,[2] that when the thyroid is mutilated, changes similar to those to which Greenfield has referred, regularly occur. In this case we are dealing with a regenerating gland-stump, but Canizzero[3] has found closely similar changes to occur as the result of slight injuries to the gland, such as were induced by separating it carefully from its attachments for the purpose of uniting it to the muscles of the neck. The histological changes occur-

[1] Operation by Dr. J. C. Warren. See a paper by the writer in the *New York Journal of Nervous and Mental Diseases*, December, 1893.

[2] Unpublished communication to the Association of American Physicians; see *Transactions* for 1893.

[3] *Deut. med. Wochenschr.*, 1892, p. 184.

ring under these circumstances are characterised as a reversion towards the embryonic state. Perhaps they indicate an attempt at more active functionating, but more evidence of this is needed than we now possess. It is also noteworthy that even in phthisis changes perhaps analagous may occur.[1]

Again, it sometimes happens that Graves' disease gives place rapidly to myxœdema,[2] and this certainly looks as if the changes in the gland in Graves' disease were of a destructive tendency. Moreover, it is well-known that Graves' disease sometimes occurs gradually as a complication of ordinary goitre, and we have no evidence to show that under these circumstances any further increase in the secretion of the gland goes on.

Furthermore, if we may trust an observation by Sollier,[3] myxœdema, beside its liability to follow Graves' disease, or to occur in some other member of the family of a goitrous patient, may even co-exist in some measure with it.

Yet myxœdema can hardly be supposed to come on while the thyroid is secreting an excess of its active principle.

It is true that some of the symptoms of Graves' disease suggest the presence of some poison, and physiological experiments with the urine of such patients seem to indicate an unusual toxicity,[4] but we know nothing of the origin of this poison, nor whether it is the cause or result of the nerve disease.

It would be difficult to explain, on any toxic theory, the fact that the exophthalmos is usually greater on the side of the larger lobe of the thyroid gland.

Whether the thyroidal theory stands or falls, there is another element in the causation of Graves' disease which is so important that we ought to hold a clear conception of it in our minds. Before speaking of this, however, I shall

[1] Defaucomberge, cited by Horsley, *British Medical Journal*, vol. i., 1892.

[2] See Clinical Society's Report, 1886, and a paper by the writer published in the *American Journal of the Medical Sciences*, September, 1893.

[3] *Rev. de med.*, 1891, p. 1000.

[4] See for example Boinet et Silbert, *Rev. de med.*, 1892, No. 1.

call your attention to a few peculiarities in the history of Graves' disease, and of the patients who are afflicted with it. In the first place, it is admitted that Graves' disease often appears, and often with great suddenness, under the influence of fright or other forms of painful emotion. These are, in fact, its most common exciting causes. It is not, however, generally recognised that the symptoms may also disappear with considerable rapidity, in an acute case, provided the conditions are favourable. Dr. Gowers reports such a case, and my colleague, Dr. F. Coggeshall, has seen a very striking instance. Again, Graves' disease rarely makes its appearance except in neuropathic families. Finally, I wish to point out the very striking fact that Graves' disease may be complicated with any one or more of a variety of other disturbances of the nervous system, and finally of other organs of the body. Such accompaniments are various forms of insanity, diabetes, muscular atrophy, and even myxœdema.[1]

The bond between these two affections may be analagous to the bond which unites Graves' disease to so many other morbid processes; that is, the existence of a neuropathic taint. Generally, to be sure, myxœdema is associated with Graves' disease, and occurs when the destructive processes which occur in the gland have reached a certain point.

There is one important principle which should be kept clearly in sight when we endeavour to trace out the ætiology of an important nervous affection like the one now before us, and this is that the symptom complexes which we see in the disease are often only exaggerations or caricatures of physiological or quasi-physiological arrangements. Thus, there is the physiological provision for rapid action of the heart to meet the needs of sudden exertion. But when this arrangement works, as one might say, too well, *i.e.*, over sensitively, we have morbid forms of palpitation. Similarly, the physiological act of attention in the presence of danger includes fixation, and perhaps even slight protrusion, of the eyeballs, and a general tension of the muscles of the body, and this often passes over into something closely resembling the symp-

[1] Sollier, *Rev. de med.*, 1893.

tom complex of Graves' disease. The fixed muscles begin to tremble, the heart beats come quicker, the breath comes faster, watery discharges may occur, and the whole vasomotor system is liable to be more or less paralysed.

If all cases of Graves' disease originated in fright, we might well assume that the symptoms were due to an overstrain of medullary and other centres intimately related to the emotional centres of the brain. But even for a case not originating in fright this explanation may be made to throw light on the origin of the symptoms. It is evident that there are a set of functions, the activity of which is closely related to emotional excitement, and which form a more or less definite group. What we have to assume is that this group of correlated functions or the nervous arrangement which underlies them, form one of the weak points of the nervous system. When the nervous system as a whole suffers a severe strain, this is one of the places through which the line of fracture, so to speak, is liable to run, just as under other circumstances it runs so as to produce progressive muscular atrophy, or epilepsy, or hysteria; and when either of these latter disturbances occur in connection with Graves' disease, it is probably because the nervous system of the individual concerned has given way simultaneously in more than one direction. Usually, when the nervous system gives way at one or another point, it is possible to find some locally acting cause which made that point especially weak for the time being. This may be one of many relating causes, such as an inherited local weakness, an exceptional local strain, or, in the case of Graves' disease, local irritation of various sorts, or a temporary paralysis of the central vasomotor system. It is to the great merit of the French school to have enlightened us on these various disturbances which form what they call *the neuropathic family*, and with skill and clearness to have pointed out the relation between them. Raymond and Serieux have done this for exophthalmic goitre[1] in an interesting and valuable paper. If the lines that they draw between the different nervous

[1] *Rev. de. med.*, 1892, No. 12.

affections to which they refer are too sharp, we may pardon the error for the sake of the practical value of the picture which they present. The opinion of these writers is essentially that which I have thus attempted briefly to state. They believe, that Graves' disease may be best conceived of as a disorder of the nerve arrangements in the pons and medulla, which enter into activity as a part of the emotional expression. When to this group of phenomena other nervous symptoms are added (mental symptoms, &c.), or signs of an exaggeration or giving way of other nerve activities, they consider that we have to deal, not with additional symptoms of Graves' disease, but with a series of more or less independent affections, co-existing, because all are similarly expressions of one morbid influence, namely the neuropathic state. These writers quote Peter[1] as pointing out a resemblance between the symptoms of Graves' disease and those of strong emotional excitement. But so far as the priority of conception is concerned, this view was advanced by an English writer a number of years ago (reference now unknown to me). In this connection I refer also to Darwin's work on emotion in animals. For that matter the idea is so striking that it has doubtless suggested itself to many minds independently. It is certainly a merit of the French conception of the tendency of these neurotic manifestations to show themselves coincidently that it helps us to understand the diversity of the clinical pictures presented by different patients suffering with Graves' disease. The idea would be that as the nervous system breaks down in one direction, this very fact is liable to render it more prone to break down in others on account of the excesses and excitations to which the first sickness gives rise.

There is one other mode of view of the pathogenesis of Graves' disease to which it is important that a few words should be given. This is the conception of it as due in part to excitation of the nerves coming from the vagus and sympathetic, which ramify so plentifully in and around the thy-

[1] *Bull. med.*, April 23, 1890.

roid gland. This view is ably presented by Wette[1] on the basis of a large experience with the surgical treatment of goitre of all sorts. A somewhat similar position is taken by Mueller.[2] Both these writers believe that the nervous symptoms which are so common as an accompaniment of ordinary goitre pass over by insensible gradation into those which are characteristic of typical Graves' disease, though Wette inclines to the view that in the latter case symptoms are superadded, which he considers to be probably due to toxic products from the affected gland. Two of the cases of ordinary goitre reported by Wette are especially important, because they indicate how ordinary goitre is liable to affect the heart independently of the symptoms of Graves' disease. These are cases, namely, where the heart's action seemed to have been *slowed* by the irritations starting from the goitre, and we can imagine that this might happen often were it not that, as he says, the inhibitory arrangements on which this slowing depends are always the first to suffer under over-stimulation.

It is, indeed, improbable that the thyroid gland, with its rich nerve supply, should swell and pulsate and become filled with masses of new connective tissue without giving rise to nerve excitations, especially in view of the fact, to which I have already alluded, that irritations in the nasal cavity seem to act as the source of so many reflex symptoms, among which are occasionally exophthalmos and perhaps other of the signs of Graves' disease.

To sum the whole matter up, we have two questions to bear in mind as regards the ætiology of Graves' disease: First, what is the underlying predisposing cause? Second, what is the mechanism by which this cause acts? The underlying cause I believe to be an undue excitement of the quasi-physiological arrangement called into play frequently as a part of emotional excitement. This arrangement represents the channel into which emotional excitement naturally pours itself after rising to a certain height, and its

[1] *Arch. für Klin. Chir.*, p. 44, 1892.
[2] *Deut. Arch. für Klin. Med.*, 1893.

anatomical centre is in the medulla oblongata, and related parts.

As regards the modes in which this nerve arrangement underlying the symptoms of Graves' disease is actually disturbed, they may be many, and among them there seems it may be that poisoning by the thyroid secretion is one active agent. This is, however, a matter of some doubt, because we have no right to assume that if the symptoms of Graves' disease are habitually due to the toxic action of the thyroid secretion, they are still capable of being excited in the same form without the aid of that secretion, and if we can get, as it appears, the greater part of these symptoms under quasi-physiological conditions, then it would seem unnecessary and somewhat gratuitous to invoke the thyroid secretion as an habitual cause of them in disease. Neverthless, it would appear that the physiological action of the thyroid extracts does excite a series of symptoms somewhat resembling those of the affection with which we are dealing.

[The exhaustive, historical and analytical monograph on Basedow's disease, by Leopold Hirsch (*Sammlung Klinischen Vorträge*, 1894) came to my notice after this paper was prepared, so that I have not attempted to utilise it fully. The author's careful analysis makes it clear that the time is not ripe for accepting any exclusive theory of the causation of the disease before us.]

As regards the *treatment* of Graves' disease, the following facts are to be borne carefully in mind: First, in the early stages of the disease it is often possible to effect an improvement or cure by the removal of the exciting cause, combined with the use of such influences as will secure the most absolute physiological rest to the nervous system. Occasionally chlorosis seems to act as such an exciting and removable cause (Robertson); at other times the irritation of some certain nerves; while in other cases the only obvious factor has been a strong emotional excitement which needs to be counteracted by profound quiet, combined with tonic treatment.

Second, one cannot review the history of the therapeutics of this affection carefully without becoming con-

vinced that a long step toward recovery is made if one can bring to an end any one of the series of partial causes which are tending to maintain the disease. It is on this principle that we can explain the beneficial effect of the treatment of the nasal cavity, the removal of the thyroid gland, and the measures specially designed to quiet the action of the heart. The fact that by partial thyroidectomy we are able to remove a certain amount of secreting tissue of the gland, does not, I think, at all prove that the thyroid disease was the principal or only cause of the affection. What we probably accomplish in that way is the removal of a part of the excitations which combine to keep all the unstable nerve centres in an irritable state.

The records of the result of operation on the thyroid seem very favourable so far as they have been reported. Out of more than fifty cases there have been three or four deaths, and in almost all the rest a greater or less degree of improvement has resulted, frequently a substantial cure. Nevertheless, the operation should count as a dangerous one, especially in cases of weak heart. In two cases observed by myself,[1] however, the results have not been so favourable as I had been led to hope. One patient died at the end of a week, though apparently doing well. The other patient is better than before the operation, though by no means well. The thyroid at first diminished in size, but is now increasing again. I have also seen a case where a relapse occurred two years after the operation.

It happens not infrequently that an operation is followed by paresis or paralysis of one recurrent laryngeal nerve. This happened in my first case, and the paralysis has not passed away, though the voice has returned. An operation has been devised to prevent this, which consists in leaving the back part of the gland with its attachment, and removing the anterior half (Miculicz).

It is very desirable in making further studies of the effects of the operation that surgeons should pay special attention to the symptoms of the first few days after the

[1] *New York Journal of Nervous and Mental Diseases*, December, 1893, and May, 1894.

operation, in order to test the correctness of the theory which I have advanced that a patient suffers from thyroid poisoning during that period.

The indications for and against surgical operation have been very recently, though briefly, discussed by Rehu.[1]

He thinks that operative treatment of the goitre in one or another way is the treatment *par excellence* for Graves' disease, but that if thyroidectomy is undertaken in advanced stages of the disease, or when the heart is excessively weak and irritable, it is attended with great danger. Two of his patients died from cardiac failure shortly after the operation, and it is noteworthy that in one of these cases the operation consisted only in ligaturing the thyroid arteries, so that there is no reason to accuse thyroid poisoning as having been the cause. Another patient died of pneumonia, and a fourth died on the operating table from the effects of tying the stump of the gland to check hæmorrhage. Rehu has seen benefit, in light cases and in early stages of the disease, from injections of iodoform, and recommends ligature of the arteries for very vascular goitres.

As a rule, however, he thinks resection is the best method, and that it should not be delayed until the patient is *in extremis*. Personally, I do not feel that the last word has been said with regard to the dangers of the operation or as to the means of anticipating or avoiding them.

As regards the drugs which have proved of benefit, the two most important are probably iron and belladonna. It has been suggested that belladonna acts by diminishing the secretions from the diseased thyroid gland, but it is also well known to quiet the heart in certain conditions independently of the other mode of action. I have not seen much benefit from its use. Picrotoxin was recommended by Bartholow, but Robertson tried it without any effect. Ergot occasionally seems to be useful, and iodide of mercury ointment, or some other form of iodine ointment, seems to act beneficially in reducing the size of the enlarged thyroid gland. Mental and physical rest are, however, besides thyroidec-

[1] *Loc. cit.*

tomy and the treatment of the chlorotic and anæmic conditions, undoubtedly the most effective.

A patient of mine who had been severely ill for many years began to improve the moment when she commenced to lie in the open air many hours daily, and this practice she continued with benefit both winter and summer.[1] Eventually the thyroid, which had never been more than moderately large, practically disappeared, the heart's action became almost normal, and the exophthalmos much less, so that the patient has returned to moderately active life.

[1] See also, J. Madison Taylor, *Medical News*, December, 1893.

PERIPHERAL NEURITIS IN EXOPHTHALMIC GOITRE.

BY ARTHUR MAUDE.

In the paper I read at the opening meeting of the London Medical Society in October, I endeavoured to produce evidence, purely clinical, that peripheral neuritis was a common epiphenomenon in exophthalmic goitre. In the discussion which followed, the only notice which was taken of what was at any rate a novel point, was a direct statement from a well-known neurologist that he did not see any reason for supposing that neuritis was ever present. May I, therefore, ask to submit my views to a more highly trained audience of neurologists in these pages?

I was at the time quite unaware of Professor Greenfield's late researches in the microscopical anatomy of exophthalmic goitre;[1] and it is premature to criticise them in detail, as they are still incomplete. I pointed out, however, that no examinations, after modern methods, have yet been made of the peripheral nerve trunks; and I still look to such examination to show changes comparable to the so-called multiple neuritis produced by various toxic agents.

The symptoms I ground my supposition on are:—

(1) The frequent occurrence of "cramp." Dr. Hector Mackenzie,[2] who first drew attention to it, found it present to some degree in thirteen out of fifteen instances, a proportion I can verify from my own experience. These painful contractions often assume the form of veritable "tetany," both of hands and feet. It is needless, since the publication

[1] "Bradshaw Lecture," 1893.

[2] *Lancet*, September 13, 1890.

of Ross and Bury's work,[1] to insist that muscular spasm of this form is due to peripheral irritation. As to the nature of this irritation two views present themselves.

(*a*) Tetany commonly follows removal of the thyroid, and often the injection of mucin into the circulation of animals (cats: Wagner and Hammerschlag).

It is possible, then, that cramps are in these cases the result of late, excessive disturbance of the thyroid, by which some poison, perhaps mucin, remains in excess in the circulation. The cases in which I have found cramp and tetany present have not all presented large goitres. But this proves nothing, for great functional disturbance may exist without great external increase of the thyroid.

(*b*) Tetany is frequently associated with disturbances of the alimentary canal, from the formation of a direct toxin, which may be separated from the digestive fluids. Precisely similar disturbances of alimentation are prevalent in Graves' disease. In the few cases in which I have been able to follow out this point, cramps were always associated with gastro-intestinal troubles.

(2) Ordinary sensibility is nearly always increased: "Hyperæsthesia is as universal in the sensory as tremor in the motor nervous mechanism" (Grainger Stewart[2]).

(3) Symmetrical paresis of the legs is the rule. It rarely amounts to complete paraplegia, and is never associated with rectal or cystic paralysis.

(4) The patellar reflexes are nearly always lessened or entirely abolished (Marie): this is my own experience also. On the other hand, Grainger Stewart states that the myotatic irritability varies much, that "the knee-jerks may be sometimes increased, and ankle clonus even be present, more especially in the cases manifesting a high degree of nervous excitability, but on the other hand, the knee-jerk may be entirely absent."

(5) I find varying degrees of numbness, tingling and pains in the peripheral nerves of the legs, a common cause of complaint.

[1] "Peripheral Neuritis," Ross and Bury, p. 398, 1893.

[2] *Edinburgh Hospital Reports.*

(6) I would suggest that those forms of localised œdema evidently of neuropathic origin, so frequent in Graves' disease,[1] are, in fact, manifestations of peripheral neuritis; comparable as they are to the conditions described by Dr. Bristowe[2] as hysterical neuritis, in which œdema was occasionally a prominent feature. The comparison is also striking between these localised œdemas and those also seen in beri-beri, which is clearly a "peripheral neuritis."

We have here, I think, all the clinical attributes of peripheral neuritis in an early stage, but in that stage it seems to stop. I have seen no cases, in record or in my own practice, which point to advanced neuritis. And as this arrest is also a feature of the neuritis of diabetes, I have examined all my cases carefully for glycosuria, with negative results. On the other hand, so many of these patients present gastro-intestinal disturbances that it is possible that the irritating poison may be of alimentary origin; while it may be some substance formed in the thyroid, or which should have been eliminated there.

Dr. Judson Bury informs me (August 29, 1893) that he remembers one case in which in addition to the ordinary tumour there was the most marked fibrillation of the interosseous muscles of the hand. This he adduces, quite correctly, as evidence of peripheral neuritis. He kindly adds: "I quite agree with you as to the probable association of peripheral neuritis and Graves' disease."

The point is, perhaps, worth following out by neurologists, both at the bedside and by microscopical examination."

[1] Maude, *Practitioner*, December, 1891.
[2] *British Medical Journal*, 1892, ii., p. 1098.

A CLASSIFICATION OF REFLEX ACTIONS.

BY D. FRASER HARRIS, B.SC. (LOND.), M.B., C.M. (GLASGOW).

THE accompanying classification is an attempt to exhibit in one view a scheme of the various groups of the reflex actions of the animal body arranged in ascending degrees of psychological complexity (*vide* column B.)

Both physiological and pathological reflexes are noted, though it was possible to quote only one or two examples of each subgroup out of a very large number.

The scheme aims at providing a more extensive and at the same time more definite terminology than is at present in use, whereby it would be possible to refer, with considerable precision, any given reflex to its appropriate genus or species.

The classification is more an indication of the lines on which such could be carried out than any final or rigid presentation of the question. It has however, I think, a certain naturalness and symmetry in its construction.

A terminology that simultaneously recognises the psychological and physiological aspects of the same action has its advantages.

We might include under "excito-motor," all those obviously simple or "low" reflexes which (*a*) do not require consciousness even for their existence; (*b*) do not affect it (*i.e.*, the majority of them are outside its "pale"); and (*c*) are not able to be voluntarily controlled (or "inhibited").

They are, for the most part, the actions, movements and processes of "vegetative" or "organic" life.

If, however, such a reflex affect consciousness, or be in any measure, voluntarily controlled, then it rises *psychologically* into a higher category, while its physiological nature has undergone no change.

Thus euperistalsis, conforming to *a*, *b* and *c*, is merely excito-motor; but when consciousness becomes affected as

in dysperistalsis (colic), it is an algio-motor reflex—psychically higher. Again the unconscious micturition in a sleeping child is excito-muscular, whereas healthy adult micturition is sensori-muscular, consciousness being affected, and the reflex (within limits) being capable of voluntary control—physiologically they are quite similar so far as the mechanism of the act is concerned. Intermediate in psychical value between mere stimulations and the sensori-motor group are the algio-motor reflexes, for which consciousness at least of pain is a *sine quâ non*.

Under group 3, the sensori-motor, are included all those reflexes whose afferent currents have originated in a true *sense*-organ, there having existed a perception (to be strictly accurate) between the reception of the afferent current and the emission of the efferent. Now this, like most *natural* groups, has no rigid confines, for, in the first place, if the sensation be painful, the pain may be *the* predominating mental state, and the subsequent reflex be an algio-motor; while, on the higher side, it merges into group 4 or 5, both emotions and ideas being pre-eminently stirred up by sensations *pre*sented (*re*-presented sensations being "ideas" themselves).

But when an emotion (however produced) is the *characteristic* of the mental state *causally* preceding a reflex, that reflex is emotio-motor, the emotion being *the* intermediary between the afferent and the efferent currents. Almost no state is purely perceptive, emotional or ideational, yet it may be one of these *characteristically*, and *as such* gives rise to *its* reflex.

The ideo-motor group includes all reflexes which are not the result of either perception or emotion; in some sense "residual phenomena" among reflexes.

The "ideas" may, of course, be perceptions or emotions *re*presented.

It would be under the heading "ideo-muscular" that one would classify "volitions" if disposed to do so—a proceeding very acceptable to certain advanced psychological physiologists. And yet, to call all voluntary acts reflex, seems to lose sight of certain most radical distinctions be-

tween sets of actions, most notably between a reflex act (as we all understand it), and what one might call the corresponding voluntary *imitation* of it, *e.g.*, winking from a strong light is a sensori-muscular reflex; winking *voluntarily* is *psychologically* a process of a totally different kind.

The notion of a "reflex," as a thing done in "spite of a person," vanishes. Again, laughing "in spite of yourself," is an emotio-muscular reflex (afferent currents being from ear or eye or both), whereas voluntary laughing, *i.e.*, imitation of laughing, is, on the face of it, a very different act. Of course, many reflexes can be controlled by the will, but to identify an action which is habitually uncontrolled by the will with one which must be both originated and controlled by the will seems confusing.

The will naturally suggests "reflex inhibition." This is largely, but by no means entirely, voluntary.

I suggest that a reflex action (as ordinarily understood) be designated + (positive), and that when "inhibited" or prevented from taking place (either by the restraining influence of a higher centre upon the special "reflex" centre in question, or by the advent of an impulse to the lower centre which prevents that discharge) it be called a negative (—) reflex. Under this last category might be included reflex inhibitions of acts other than reflex themselves.

Thus the will notoriously both inhibits reflexes (as in the postponement of adult defæcation) and reflexly inhibits; but a powerful "idea" can as efficiently reflexly inhibit, as when a sudden thought renders a person speechless for the time being (*negative* ideo-muscular reflex).

It is almost superfluous to add that an emotion can inhibit reflex action, as when "fear" causes dry mouth (*negative* emotio-glandular reflex), or when "joy" produces syncope (in this case a term is already in use—cardio-inhibitory reflex).

Lastly, pain can reflexly inhibit, as in syncope from "shock" from operation (negative algio-muscular reflex on cardiac muscle).

As to "vaso-motor" reflexes, if there be but *one* centre "vaso-motor," whose function it is to maintain general

vascular tonus, then active (arterial) hyperæmia, due to inhibition of the centre, would be a negative excito-vascular reflex ("vaso-inhibitory" of authors), while pallor would be the positive excito-vascular reflex ("vaso-constrictor"). If there be *two* centres, one for vaso-constriction, Vc., and one for vaso-dilatation, Vd., then, *e.g.*, blushing from present emotion would be either: A + emotio-vascular reflex through Vd., or A — emotio-vascular reflex through Vc.; and similarly, emotional blanching would be either: A + emotio-vascular reflex through Vc., or A — emotio-vascular reflex through Vd.

A word as to psychico-metabolic reflexes. Of course, all vital action is "metabolic," but one can distinguish a kind of reflex concerned, not with definite glands or vessels, but having its manifestations through a wide spatial distribution.

We may, as before, have metabolism stimulated or depressed, and thus a + psychico-metabolic reflex might be more shortly named psychico-anabolic, while a — psychico-metabolic reflex would be a psychico-katabolic reflex.

Thus, the hair turning white through emotion would be a negative emotio-metabolic reflex.

The emotional state has acted "*reflexly*" on centres trophic to the nerves influencing the formation of pigment over a considerable area of skin.

It would be under the category "*ideo*-metabolic" that we would place those well-authenticated cases of hallucinations causing or curing disease. Thus there is much mystery and romance dispelled when instead of "miraculous cure by faith-healing" we write "positive ideo-metabolic (systemic) reflex!" Of course, metabolic reflexes might exist in other systems than the dermal.

A final word on reflexes. It will be very evident that "expression of the emotions" is largely reflex, while any imitation of an emotion—simulation of a particular expression—is a different thing psychologically; it is voluntary to begin with. Moreover, expression of many emotions is eminently under control: self-command does not so much consist in not experiencing certain emotions, as in being habitually able to "inhibit" or control their manifestations.

A CLASSIFICATION OF REFLEX ACTIONS ON A PSYCHICO-PHYSIOLOGICAL BASIS.

A	B	C	D	EXAMPLES.
I. PERIPHERO-MOTOR.	1. *Excito-motor.*	Excito-muscular	Of Involuntary muscle.	Euperistalsis. Micturition in sleeping child. Early non-voluntary stages of Parturition. Defæcation and micturition in "spinal disease" and in coma. Iris reflex in partial narcosis. Hiccough. Ejaculatio seminis during sleep. Later stages of deglutition.
			Of Voluntary muscle.	Spasm in children during sleep from any irritation, *e.g.*, worms. Movements of unconscious adjustment in sleep.
		Excito-glandular.		Outpouring of gastric and intestinal juices in digestion. "Sympathetic" preparation of the mammary glands in pregnancy.
		Excito-vascular.		Pathological blushing ("flushings") from indigestion. Erectio penis (in sleep). Probably certain tropho-neuroses, such as erythema, eczema, herpes, scleroderma, urticaria, "glossy skin."
		Excito-metabolic (Dermal).		Pigmentation of areola in pregnancy. Chloasma in disease.
	2. *Algio-motor.*	Algio-muscular.		Of *Involuntary* muscle: Dysperistalsis (colic). Vomiting from "renal colic," &c. Inhibition of heart ("shock") under operation without chloroform. Of *Voluntary muscle*: Spasm=writhing from pain.
		,, glandular.		Perspiration due to pain. Lachrymation from painful foreign body.
		,, vascular.		Blushing or pallor from agony.
		,, metabolic.		
	3. *Sensori-motor.*	Sensori-muscular	Of Involuntary muscle.	Contraction of iris in strong light. Action of tensor choroideæ in positive accommodation. Contraction of uterus when child is "put to the breast." Non-voluntary elements of adult, conscious micturition and defæcation.
			Of Voluntary muscle.	Coughing, sneezing. Winking from foreign body. Laughter and spasm from tickling. Spasm from draught of cold air in hydrophobia. Deep inspiration from cold affusion.
		Sensori-glandular.		Lachrymation in strong light. Salivation on eating sour or acid materials, or at sight of food.
		,, vascular.		Pallor in cold bath. Glow of skin in hot bath. Catarrh of mucous membranes (*e.g.*, nasal) from draught of air, &c.; probably with contemporaneous "low nerve tone." Rise of blood-pressure whilst hearing music.
		metabolic (dermal).		Freckles. "Bronzing" of skin in hot climates.
			Of Involuntary muscle.	Hyper-peristalsis with diarrhœa from various emotions, *e.g.*, fear. Progress of "labour" (dilatation of "os") inhibited by stranger coming in, or induced by fright.

II. CENTRO-MOTOR.	4. *Emotio-motor.*	Emotio-muscular.	Of Involuntary muscle. — Hyper-peristalsis with diarrhœa from various emotions, *e.g.*, fear. Progress of "labour" (dilatation of "os") inhibited by stranger coming in, or induced by fright. Cardio-inhibition ("syncope") from many emotions—"Heart standing still." Rate of heart beat accelerated by emotion. Micturition through emotion.
			Of Voluntary muscle. — Child jumping for joy, stamping feet from vexation, slamming door from rage. Assault and murder from violent emotion. Emotional alteration of the respiratory rhythm, *e.g.*, sighing.
		Emotio-glandular.	Activity of certain glands, *e.g.*, stink-glands of "skunk" through fear. Weeping from emotion or inability to do so. Inhibition of salivation from fear. "Cold sweat" of fear, and perspiration from other emotions. Lactation ("draught of milk") induced through joy. Eupepsia due to happy "frame of mind."
		Emotio-vascular.	Blushing from shame, modesty, rage, &c. Pallor from rage, fear, "nervousness," &c.
		Emotio-metabolic (dermal).	Hair turning grey from emotion.
	5. *Ideo-motor.*	Ideo-muscular.	Of Involuntary muscle. — Vomiting from *recollection* of disgusting sight or taste, or from vivid gustatory hallucination. Influence of hallucinations, &c., in the insane on the muscular tonus of viscera, *e.g.*, constipation of certain lunatics. Spasm of muscles of deglutition in hydrophobia at the *idea* of "water."
			Of Voluntary muscle. — Actions and co-ordinated movements in somnambulism, and the majority of such in the hypnotic trance. Laughing at a recollection. "Reflex speech" of certain lunatics. Certain so-called "secondarily automatic" actions, such as standing, sitting, walking, playing on piano (when completely familiar with it).
		Ideo-glandular.	Weeping at a recollection. Action of hallucinations (in the insane) on secretions. Salivation at thought of food.
		Ideo-vascular.	Blushing or blanching at a recollection.
		Ideo-metabolic.	Nails ceasing to grow in mania (dermal). Action of the mind over nourishment of tissue. Mentally-induced or cured diseases (as alleged). Psychically-produced tropho-neuroses.

CASE OF TUMOUR OF PONS AND LEFT CRUS CEREBRI.

BY SEYMOUR J. SHARKEY, M.D., F.R.C.P.

W. B., aged 43, sailor, was admitted into St. Thomas's Hospital under my care on January 22, 1892, and died on the 25th of the following May.

His family history presented no features of importance. He had had malarial fever eight years previously, but had never had syphilis. Three years ago he fell from a height of twenty-eight feet, landing on his feet and fracturing his leg near the ankle, but not injuring his head in any way.

Three months and a half ago when in the Gulf of Mexico he began to perspire so freely that it became a subject of general remark. He got rather stupid, and used to knock against objects on turning to his right. Soon after he became deaf in the right ear, and suffered from numbness all down the right half of his body, the face and head being the last parts affected.

On admission he was found to have right hemianopsia, deafness in the right ear, and paralysis of the superior rectus in each eye, and the right pupil was larger than the left. Both pupils reacted to light and accommodation, but sluggishly. There was no optic neuritis.

The right ear was completely deaf, bone as well as air conduction being abolished.

There was not only numbness over the whole right side of the body, but tactile sensation was also diminished. Sense of temperature was likewise altered, and the only difference he noticed between hot and cold things applied to the right side, was that the former gave rise to a pricking sensation. Muscular sense was normal, and there was no loss of power, except perhaps in right hand. Taste and smell were not materially altered. Superficial and deep reflexes presented no marked changes. Memory said by patient to be defective, but it was not very obviously so. Speech was rather thick, and there was a

tendency to stammering. Gait was somewhat peculiar, the right foot being raised higher than the left, and the heel striking the ground first. But this might have resulted from a partial ankylosis of ankle joint due to the old injury.

No inco-ordination or giddiness.

Profuse perspiration present, but no *visceral* disease detected.

During the first few days in hospital he suffered sometimes from bad headache, sometimes became strange in manner, and usually kept his left eye nearly closed, and the latter peculiarity remained unaltered till he died. He was, however, generally pleasant, and humorous, and much inclined to talk, but his memory got worse and some aphasia supervened.

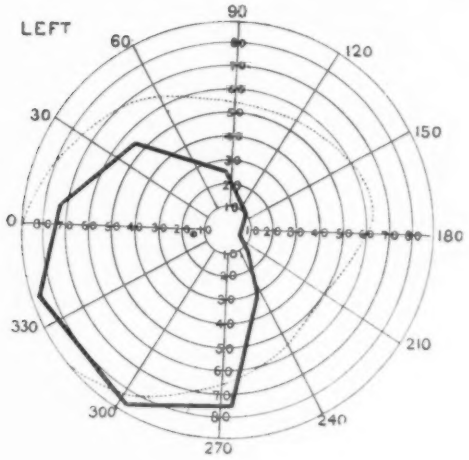
On February 1, he had forgotten the name of his last ship and captain, though he said he knew them quite well, but could not say them. When shown a candlestick and quill-pen, he was unable to name them, though he named a button correctly. When asked whether the candlestick was a watering can he became very angry, and said the question was childish and ridiculous.

On February 12 Mr. Lawford saw him and said he could read with each eye, and with one as well as the other. He could understand what he read and could write. Loss of power in both superior recti and in inferior oblique of right eye.

On February 15 Mr. Nettleship examined him and noted that there was no movement of the eyes upward beyond the horizontal, and that downward movements were also defective, more in right than in left eye. Concomitant lateral movements full, but convergence very defective, and more so in right than in left eye. Irregular nystagmus in lateral and in upward movements. Right pupil larger than left. State of accommodation doubtful. No ptosis; but left palpebral fissure always narrower than right. As to diplopia, patient appeared to say nothing about it, and he could not with any certainty be got to see double, at any rate not in such a manner as would agree with paresis of right internal rectus.

R. reads 6J. at 14″
c. + 3 D.=1J. at 9″
L. reads 14J. at 14″
c. + 4D.=1J. at 9″
Together=6J. at 14″
c. + 3D.=1J. at 9″, but prefers to shut left eye.
R. V.=$\frac{6}{12}$: Hm. 1D.
L. V.=$\frac{6}{18}$: No Hm.

Not tried with cylinders.


LEFT
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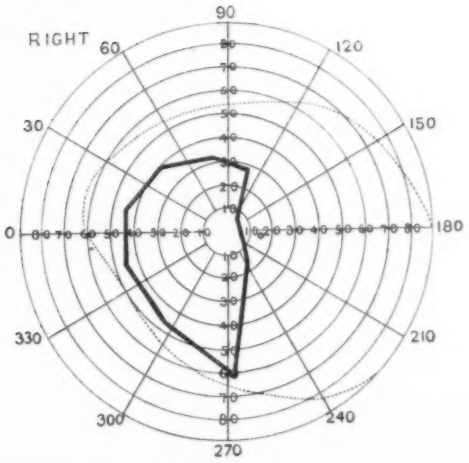
270


RIGHT
90
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180
330
210
300
240
270

Fundi normal.

Loss of memory and mental power well marked. When told to sit on his locker he could not be got to understand what was wanted; nor could he say anything he wished to say. There was no muscular paralysis, but very evident anæsthesia, as he could not feel a pin stuck into his right arm. Always yawning.

On February 27 I made the following note:—

"There is distinct loss of power in the right side of face in lower part, the left side being drawn up on laughing, the right not. Tongue protruded straight. Movements of eyes good laterally, much limited in upward direction, (?) somewhat in downward direction too. Both hands grasp poorly, right less strongly than left. Legs fairly strong, but still probably not normally so; right not quite as good as left. Knee-jerks good; no clonus. Intellect very dull at times; cannot remember the names of blotting paper, pen, &c., and speech is hesitating and stuttering. So forgetful of words is he that he cannot carry on a conversation. No fits. Discs normal."

March 5.—Mental condition varies a good deal, being much better on some days than on others. Yesterday he was found sitting up in bed eating a crust of bread which he said was a pine, which he was enjoying very much; all the time he was holding his left hand below it to prevent the juice from falling on the bed-clothes.

He was getting very drowsy. He could not now touch his nose with his eyes closed, or make his index fingers meet in the middle line, nor could he imitate movements made with his legs, though he was sometimes able after much thought to say which of his legs was uppermost.

On *March* 10 I made the following note:—

"Patient cannot read printed or written characters; he can make out a word or two here and there, but little more than that, and he appears to lack entirely the power of attention necessary for continued attempts. He cannot write. He very rarely names ordinary objects aright, and he does not seem to think it extraordinary if asked whether a book is an orange, apple, &c. Still he occasionally says a few sentences correctly. He is very good-humoured. Optic discs a little hazy, and streaked on internal border; (?) optic neuritis."

March 15.—Optic neuritis evident in both eyes. Attempt to take the field of vision had to be abandoned as he could not be got to understand what was wanted. His mental powers varied very markedly from day to day. For instance, on March 18 he named

a bunch of keys, a watch, a knife and a pen, and said he knew other things, but that his head was "mixed." On the 21st, he could not even remember his own name. At other times, *e.g.*, March 31, he called things by wrong names; a hair brush an egg brush, a comb an egg brush, blotting paper a cork.

April 7.—Patient could not now feed himself; urine passed unconsciously sometimes; great drowsiness. Right side evidently weaker than left; clonus sometimes obtained in right foot.

April 28.—In much the same condition, at times making intelligent answers, at others unable to understand what is said to him and giving irrelevant replies; sometimes unable to pronounce words properly.

May 19.—Mentally much the same, but physically very weak; unable to sit up for any length of time.

May 24.—Very drowsy; by 5 p.m. comatose with stertorous breathing, and temperature 103·4°, pulse 132. He recovered a little and the temperature fell, but soon rose again, reaching before death 106·2°, the pulse 200, and deep coma supervening.

Autopsy.—The brain was the only organ found diseased, and after a rough examination it was put aside to harden in alcohol. On looking at the hemispheres it was seen that the left was larger than the right, but the convolutions, nerves, and vessels on both sides were normal. Where the crura cerebri emerged from the pons on the under surface of the organ the left crus was seen to be much larger than the right, measuring $1\frac{1}{2}$ inch across, while the right only measured $\frac{3}{4}$ inch. The left optic tract was much flattened as it ran over the left crus cerebri.

On dividing the corpus callosum and separating the hemispheres the left crus was again seen to be very large. The corpora quadrigemina and the roof of the aqueduct of Sylvius were unrecognisable, their place being occupied by a new growth, irregular on the surface and continuous with the left crus cerebri. The whole space between the anterior and upper border of the cerebellum and the third ventricle was filled up by the tumour. The superior crura of the cerebellum could, however, be seen emerging from the tumour apparently healthy, and entering the cerebellum. The floor of the fourth ventricle viewed from above appeared to be normal. After the brain had been hardened in alcohol, sections were made.

Section 1 passed through the pons varolii vertically just beyond its junction with the medulla oblongata. The right half of the pons appeared to be healthy, but the fourth ventricle was pushed far to the right by the tumour which grew from the left half of the roof of the aqueduct of Sylvius, but the roof itself and the superior cerebellar peduncle seemed thin and healthy, as did also the greater part of the left half of the pons. The root of the tumour seemed, however, to invade the floor of the fourth ventricle.

Section 2 made vertically, $\frac{1}{5}$ inch posterior to the emergence of the crura cerebri from the pons showed the aqueduct of Sylvius pushed far to the right, the tumour involving the roof of the aqueduct and left half of pons above the locus niger (fig. 1).

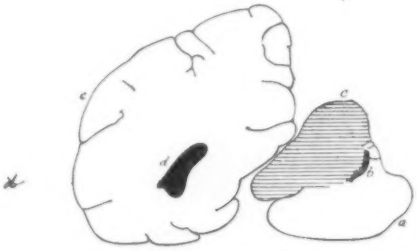

FIG. 1 (Section 2).

a. Pons varolii. *b*. Aqueduct of Sylvius. *c*. Shaded tumour. *d*. Lateral ventricle. *e*. Left hemisphere.

Section 3, made vertically through the crura cerebri about $\frac{1}{4}$ inch beyond their point of divergence, showed the right crus healthy, and the upper or dorsal part of the left entirely occupied by the tumour. The latter had obliterated the posterior part of the internal capsule, the optic thalamus, and part of the extraventricular nucleus of the corpus striatum. The hippocampal convolution was intact (fig. 2).

Section 4 just in front of corpora mammillaria. The optic thalamus, tail of caudate nucleus, and internal capsule appeared intact, the tumour appearing only in a small area

of the section and involving part of the extraventricular nucleus.

Section 5 through head of caudate nucleus just behind the anterior extremity of the third ventricle. It showed the internal capsule healthy, and slight involvement of the outer and lower part of the extraventricular nucleus.

Section 6 vertically through Broca's convolution showed nothing abnormal.

The following *résumé* of the order of appearance of the symptoms helps one to realise how easy it was to come to a conclusion as to the exact position of the tumour.

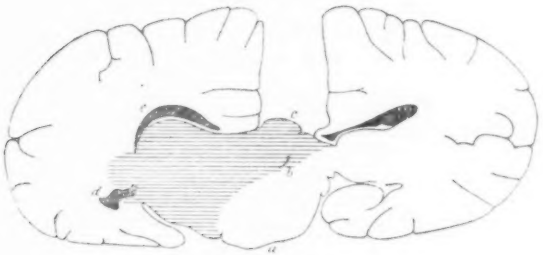

FIG. 2 (Section 3).

a. Pons varolii. *b*. Aqueduct of Sylvius. *c*. Shaded tumour. *d*. Lateral ventricle. *e*. Left hemisphere.

Three and a half months before admission:—Profuse perspiration; paralysis of superior recti of eyes (it is probable that this existed early, though only proved to exist on admission into hospital. It is not a paralysis likely to arrest the attention, and no remark was made by the patient with reference to it); hemianopia; deafness; numbness in right side.

On admission, January 22, 1892, he was found in addition to be suffering from alteration of sense of touch; alteration of sense of temperature (but no alteration of taste or smell); thickness of speech; difference in size of pupils; headache; failure of memory.

February 1.—There were found mental weakness; aphasia; utter loss of memory for words; drowsiness.

February 27.—Right facial paresis.

March 5.—Loss of taste (probably); loss of muscular sense.

March 10.—Alexia; agraphia; optic neuritis.

April 7.—Partial right hemiplegia.

May 19.—Great physical weakness.

May 24.—Coma; hyperpyrexia; death.

The paralysis of the superior recti of both sides, together with hemianopia, deafness and numbness on right side could scarcely be explained except by disease of the floor of the aqueduct of Sylvius gradually invading the left crus cerebri and internal capsule. The successive changes in the patient's condition indicated greater and greater involvement of the nerves of sense, special as well as general, and subsequently disorganisation of the more distant motor tract. It was not until late, when the tumour had reached considerable dimensions, that optic neuritis supervened.

The profuse perspiration which marked the onset of the disease, and then disappeared, is very interesting with reference to the question of the existence of sweat centres.

Another very remarkable point is the gradual abolition of mental power and of those functions which are the special attributes of the left cerebral hemisphere, owing to the cutting off of its sensory connection with the exterior, and that notwithstanding the integrity of the hemisphere itself, and that of the corpus callosum which should put it into free communication with the right hemisphere. The mental defects were very marked three months even before his death.

Critical Digest.

ON EXOPHTHALMIC GOITRE.

BY A. MAUDE.

JOFFROY[1] gives *in extenso* a series of clinical lectures delivered in 1891. He surveys carefully the whole enormous range of ascertained clinical facts, and the paper constitutes the fullest and minutest general account with which we are acquainted in any language. It is only possible to glance at some of his points. One patient, selected as a clinical example, shewed marked pulsation of the thyroid. But this is only one factor in its alteration; the purely vascular dilatation of the thyroid only exists in theory, and in books, and is never found clinically or microscopically; there is always a distinct structural change in the gland to be appreciated during life by careful manipulation; a hard consistency, usually localised and frequently nodular in distribution, these nodules being centres of inflammation or of colloid infiltration, and this conclusion is always confirmed by examinations after death if properly conducted. We do not understand M. Joffroy to deny that vascular engorgement of the thyroid does not take place frequently in healthy persons, particularly women, but that such engorgement cannot produce exophthalmic goitre. Considerable real increase may take place in the thyroid, while it remains inappreciable during life. Variations in size of the gland occur very rapidly in Graves' disease. But the point he insists upon most strongly is that the thyroid may be profoundly altered in structure and yet not increased in volume; with this we are quite in accord.

Dilatation of Arteries.—As is well known, the carotids frequently pulsate forcibly, and pulsation may be seen in the optic disc; pulsation may even be detected in the aortic arch behind the sternum. Similar pulsation is occasionally found in the ab-

dominal aorta, but the condition is rare; Charcot described it in the earliest known case in France, and Kahler reports abdominal pulsation accompanied by paroxysms of pain. We remember that Dr. L. Guthrie[2] asserted that this feature was by no means uncommon, and he sends us a note of a quite recent observation, in which the pulsation was so violent as to be visible through the bed-clothes; it ceased abruptly at the umbilicus. The patient, a woman of 60, suffered from severe epigastric pain, and gastro-intestinal crises. Rarer still are pulsations of the arteries in the arms and legs.

Photophobia is an occasional symptom, present to a marked degree in a patient of Rendu's. Joffroy found it present, but associated it in his case with paroxysmal neuralgia of the face.

He breaks new ground on the question of *Von Gräfe's symptom*. The condition has never been accurately defined even by Von Gräfe. Joffroy concludes that we must understand by the term a failure of the normally even rate of movement executed by the eyeball and the upper eyelid, and this may be disturbed in two ways: (1) By a diminution of the motility of the globe itself, which thus follows the upward movement of the lid too slowly; (2) by a paresis of the lids, which consequently move more slowly than the globes in either direction.

We are convinced that neither condition is as common as some authorities (notably Von Gräfe) have asserted; and the former condition is, we think, conspicuously rare. Joffroy offers no speculations as to the seat or nature of the lesion producing these eyelid symptoms, but it may not be out of place to consider the matter briefly. It is better, we think, to group Gräfe's sign and Stelwag's sign together as due to modification of the same cause. Von Gräfe himself ascribed the impaired movement to spasm in the "muscle of Müller," supplied by the sympathetic; and Gowers[3] regards it as caused by either spasm in Müller's fibres or in the levator palpebræ, probably the latter. We fear we cannot adhere to such an assumption of long continued spasm, unless due to paresis of opposing muscles.

The clinical facts are these:—

(1) Sometimes a *relative* over-action of the levator palpebræ (supplied by the third nerve), or of the unstriped muscle of the lids supplied by the sympathetic.

(2) A frequent paresis of the upper part of the facial muscles, the oculo-facial group (frontalis, corrugator supercilii and orbicularis).

(3) A frequent paresis of all the external ocular muscles.

Now the fibres which supply the oculo-facial group of muscles are derived not from the seventh nucleus, but from the oculo-motor nucleus, joining the seventh nerve by way of the posterior longitudinal bundle. A slight paralytic condition in this nucleus may not be sufficient to impair the movement of the globe (though it often is sufficient), but may suffice to affect the less differentiated movements of the oculo-facial muscles, and produce in them the paresis of that group, which we know does exist, or at any rate to disturb the relation of movement in those muscles to that of the muscles of the eyeball. Such cases have no connection with the facial paralysis often observed in Graves' disease. This is due to affection of the seventh nucleus or to change in the trunk of the seventh itself. The latter was certainly the case in our own patient,[4] who had Gräfe's sign before and after the general facial paralysis, which was clearly secondary to middle ear catarrh.

Ferri[5] advances the theory that Gräfe's sign is due to mechanical shortening of the levator palpebræ, from the increased volume of blood vessels distributed in the muscle substance. This may possibly be the case when the symptom has been continuous for a long period, but the irregularity and inconstancy of the condition make it impossible that such a coarse unvarying lesion should be the usual cause.

Muscular vibration of the eyelids was found in one patient; this is common enough in hysterical patients and in persons under hypnotism, and even in ordinary debilitated subjects.

Three patients under study had marked paresis of the upper part of the face, especially of the levator palpebræ; but this paresis only affects involuntary combined movement, as in suddenly directing the eyes upwards; voluntary employment of the muscle, as in intentional wrinkling of the brows, is found unimpaired. This is the reverse of the condition often found in hysterical subjects. We may remark that Vigouroux demonstrated some years ago the frequency of impairment of electrical contractility in Graves' disease.[6]

Monoplegias.—In some cases the movements of the jaw are distinctly enfeebled. Debove and Ballet described an instance of intermittent paralysis of the tongue and soft palate.

After a brief allusion to paraplegias M. Joffroy concludes the subject of paralyses by remarking that ophthalmoplegia is most common, then paralyses of the face, the jaw, the tongue, and velum palatæ; paralysis of the arms are less common and paraplegia rarest of all. This comparison is, we think, not well

founded; both from literary researches and our own personal observation paresis, and even absolute paralysis of the legs is by no means uncommon, and we think three classes of this condition may be distinguished.

(1) Cases of astasia-abasia, not of true paresis.

(2) Cases of hysterical type.

(3) Cases due to interruption of motor impulses low down in the motor tract, either spinal or peripheral. It is impossible here, however, to develop our views on this point. The paralyses of Graves' disease present a wide field of study as yet little cultivated. The difficulty is to distinguish between Graves' disease, pure and simple, and a combination with hysteria; but, M. Joffroy insists, a large proportion of "Basedowiens" do not manifest any of the ordinary features of hysteria, such as alterations in field of vision, hysterical crises, localised anæsthesias, &c.

Joffroy remarks on the abolition of patellar reflex in his patient, only to insist on the care necessary to exclude the supposition of co-existent locomotor. This, we think, is scarcely giving the symptom its true value, for Marie found it usually diminished or absent, and in investigating the point for the last few years we have found a lowered or absent reflex in nearly all severe cases.

Ophthalmoplegia Externa.—Like Ballet he distinguishes two classes of patients who present ophthalmoplegia externa: (*a*) One in which undoubted "hysterical" features are found, in whom it is impossible to assign the paralysis to either especial neurosis; (*b*) a class, less common in his belief, who cannot be considered hysterical. Paralyses of other cerebral nerves sometimes accompany ophthalmoplegia, viz., the fifth, seventh, and even the twelfth (Jendrassik).

(For bibliog. on this subject *v.* BRAIN, part lvii., vol. xv., p. 121.)

Cutaneous Rashes.—After a review of the already well-known forms of eruption or changes in pigmentation, he describes a double form of rash to us hitherto unknown. This consisted of an eruption appearing on the feet and legs resembling the later stages of the rash of measles, and mixed with this appeared a few days later a patch of small purpuric spots. This form of rash is, we say, hitherto undescribed, but by a coincidence, almost incredible, and well worth the consideration of the Psychical Research Society, on the same evening that we were reviewing this part of M. Joffroy's paper a patient of ours developed this rash suddenly.[1] She came to us two days later; there

were then a few patches of purpura on the cheeks and one elbow, and over the lower part of the face and neck a large crop of raised dusky papules, resembling measles; this was accompanied by pain over the seat of distribution of the rash. The spots faded and were succeeded by a second crop of papules a week afterwards. This patient is the subject of previous goitre, and the daughter of a myxœdematous mother.[8]

Tetany.—M. Joffroy corroborates the observations of Dr. H. Mackenzie as to the frequency of spastic contractions of the hands and feet, and inclines to the opinion that they are of thyroidal origin, similar in fact to the tetany, which may supervene after removal of the thyroid.

Muscular Atrophy.—In most cases of paralysis there is no impairment of muscle nutrition. Vigouroux has described a case in which atrophy accompanied paraplegia: Dreyfus Brissac, Du Cazal, Hüber and Möbius give similar examples. Joffroy remarks that with the exception of Hüber's case, in which the occurrence of reaction of degeneration was noted in the affected arm, the cases are insufficiently reported, and there is no note as to electrical reactions.

Psychosis.—M. Joffroy's pupil, Raymond Martin,[9] collected twenty-eight cases of mental aberration in exophthalmic goitre; he lays great stress on the variety of mental changes found, and deduces the conclusion that these changes are the outcome of disturbed cerebral involution, coupled with hereditary predisposition to psychic abnormality; that there is not, in fact, any definite type of amentia, and that the cardio-vascular condition is insufficient of itself to produce change in a normal brain. We are uncertain how far this paper is inspired by M. Joffroy himself; probably to no great degree, for in a previous paragraph the professor sums up shortly the prevailing mental changes in Graves' disease, which, though they rarely amount to mania, are yet very distinct psychoses; viz., a constant restlessness, variability of temper, with alternations of excitement and depression. This alternation is very marked, and all the more marked because, though acute mania is by no means uncommon, true alternating mania (the "folie à deux" of the French) is almost unknown.

Joffroy and Achard[10] give a full account of six cases with *post-mortem* examinations of each. In five of them the thyroid was carefully examined histologically; it is only during the last few years that this has been done, and the observations in this paper are fully confirmed by the fine series of sections exhibited

at his Bradshaw lecture by Professor Greenfield (1893). Joffroy's cases showed that even when the thyroid is not demonstrably enlarged during life, it was in all cases but one found enlarged after death, and in this one instance, though not enlarged, it was profoundly altered. We laid great stress last year on the fact, we believe undeniable, that all cases of Graves' disease, if properly watched, do show variations in the size of the thyroid, a turgescence similar to that of other glands during active secretion.

(2) The thyroid presents great diversity of change in all these cases. In one there was marked cystic dilatation of the vesicles. In three others the most important feature seemed to be the disappearance of the colloid material in the vesicles, or its replacement by small round cells, a condition we may regard as a catarrhal proliferation of the vesicular walls. In one instance there was a considerable formation of new adenomatous cell tissue, while the interstitial trabeculæ had undergone more or less fibroid degeneration, and this may be accompanied by œdematous swelling and infiltration of the fibrous tissue by leucocytes and interfibrous hæmorrhages.

Joffroy remarks that there is no hard and fast line of distinction between the condition of the thyroid in exophthalmic goitre and simple bronchocele, and he claims that this is not unlikely as so many cases are now on record in which apparently simple goitre has been followed by symptoms of Graves' disease. This view is quite in accord with our own, and to it we shall allude again. He raises the question whether the varieties of co-existent changes in the thyroid do not imply that the whole symptom-complex is in reality produced by an alteration in secretion in that gland dependent on one of several structural alterations.

We were, ourselves, impressed by the idea, on examining Professor Greenfield's specimens, that the various changes demonstrated (destruction of colloid, cell proliferation, adenomata, cystic and finally fibroid degeneration) were truly stages in an alteration beginning in catarrh, and ending in fibrosis or cystic degeneration, comparable to the same range of changes seen in the ovary or lung.

These six cases show a high proportion of concomitant distinct nervous conditions, viz., hysteria (1, 4 and 6), syringomyelia (3), locomotor (4), melancholia (2). The sixth case presented typical myxœdema supervening on Graves' disease. As the myxœdema advanced the goitre and exophthalmos, both previously well marked, subsided. As to the relation of the two

conditions, M. Joffroy w... not commit himself definitely, but he inclines to the belief tha: the coincidence is not accidental, but that in such cases (which are now not rarely known) an early catarrhal, or proliferatio: stage, in the thyroid, which is accompanied by the symptoms o: Graves' disease, is gradually succeeded by progressive filroid degeneration of the gland, which, of course, produces myxœdema. We fail to see how this view can be disputed. In his myxœdematous patient the thyroid had undergone complete fibrosis, a few small cellular masses representing all that was left of the vesicles. The examination of the nervous system in all these cases was exhaustive, and disclosed these points.

Case 1.—Nervous system showed no changes.

Case 2.—The only change found was some Wallerian degeneration of the left vagus.

Case 3.—Syringomyelia.

Case 4.—Typical tabes dorsalis.—We review this case in detail later on.

Case 5.—The cervical spinal cord showed some sclerosis of Goll's columns, and columns of Burdach. There was no lesion in the restiform bodies.

Cases 1 and 4 both showed degeneration in several peripheral nerve trunks. We wish to draw attention to this as confirming our own observation that clinical symptoms of peripheral neuritis are by no means rare in Graves' disease.[11] Greenfield also observed changes scattered all over the nervous system, which (from their nature and distribution) resemble those produced in tetanus and hydrophobia, and suggest their dependence on an irritative toxic action.

O. Hezel[12] gives a full and minute account of a *post-mortem* examination of a typical case, a woman aged 45. The microscopic examination was full, and all the details of staining are given, so that we are able to say that the best methods were used. The medulla, sympathetic trunks and ganglia, the vagi, the greater number of cerebral nerves, the thyroid and muscular substance of the heart were all carefully investigated. No changes were discovered in the restiform bodies or solitary bundles. The changes in the cervical sympathetic ganglia were marked, and though limited in extent, affected portions of both sides. The main changes were :—

(1) Infiltration by round cells of the walls of the small vessels, with occasionally narrowing of their lumen.

(2) Small hæmorrhages.

(3) Nuclear proliferation of the connective tissue capsules of

the ganglion cells, often coupled with round cell infiltration in the neighbourhood.

(4) Localised degeneration of the interstitial connective tissue, often accompanied by round cell infiltration.

(5) Degenerative changes in a number of ganglion cells, exhibiting different stages of atrophy.

The conditions described here are exactly those found by Professor Greenfield in the two cases in which he has examined the sympathetic "by modern methods;" and there can be no doubt, as he says, that the sympathetic ganglia are the seat of a subacute inflammatory process. We must not, however, be too ready to accept this explanation in the face of the fact noted by Hale White[13] that the infiltration of sympathetic ganglia, apart from Graves' disease, is no uncommon feature.

The cardiac muscle was carefully examined with negative result, though there was obvious cardiac failure during life. The thyroid was considerably enlarged.

There was striking proliferation of the epithelial cells in many of the acini. The cells stand crowded together and have a cylindrical form. Many of the cells are increased in size, and the nuclei are much enlarged. The acini are frequently stuffed with epithelial cells, so that the resemblance to the catarrh of a mucous surface is at once suggested. The whole change, however, resembles not so much a simple hyperplasia as an adenomatous condition. The whole description, we should say, tallies with that of Prof. Greenfield's; the change of epithelial lining from a cubical to a columnar form, the increased proliferation similar to that seen in adenomata, and the masses of cells lying in the spaces, are described exactly as in the late Bradshaw lecture.

Professor Thomson[14] reviews eight consecutive cases in his private practice. He dismisses fright and nervous shock as a true factor in causation, being much more often an accidental event in the course of a chronic malady which seems to date its beginning. He remarks that simple irritation of the glosso-pharyngeal and superior laryngeal nerves is sometimes accompanied by general muscular tremor, and supports this by an example from his own practice, when the impaction of a fish bone caused such tremor, while its removal at once stopped the symptom. From this he argues that the tremor of strumipriva, which occurs early after the operation, is the result of direct surgical injury to the superior and recurrent laryngeal nerve branches. But we must point out, that this is ignoring the fact that tremor occurs also in the early stages of idiopathic myxœdema,

and to argue thus from one instance where an alarming accident had suddenly occurred, with its attendant terror, is premature.

He then reviews, only to discredit them all, on the ground of their inconstancy, the various described lesions found in Graves' disease, and the experiments of Filehne, Seeligmüller and Bienfait. He details the progress and relapses of a patient, the subject of severe diarrhœa, in whom alternate koumiss and meat diet were pursued; the meat diet invariably produced severe relapses, ultimately fatal. He lays great stress on the frequency of diarrhœa in Graves' disease, and seeks to prove that the whole condition is due to an over-stimulation of the thyroid by some toxin, formed in the intestines from the decomposition of nitrogenous foods. Great stress is accordingly laid on the value of fermented milk, sold in New York under the name of "Matzoon." He recommends treatment by intestinal antiseptics, such as β naphthol and ichthyol. The whole paper is very suggestive and practical; and we have pointed out elsewhere our own belief that certainly some of the secondary features of this disease are due to intestinal auto-intoxication, though we are unable to follow Dr. Thomson along the whole line of his belief.

MM. Joffroy and Achard[15] discuss fully the coincidence of exophthalmic goitre and locomotor ataxy; they give an account of a patient, a woman of 49, in whom the symptoms of the two disorders progressed side by side. Ataxia was first discovered in 1879 and proptosis was then present. In 1885 the inco-ordination was marked, and muscular sense quite absent; the reflexes were abolished, there was typical swelling of the left knee, with subluxation of the joint and destruction of the internal condyle; Argyll Robertson pupils were present. The pulse rate was rapid and marked exophthalmos existed. She remained in a stationary state while under observation, and eventually died in 1891 of pulmonary tuberculosis.

A *post-mortem* examination was made, disclosing tuberculosis of both lungs. The thyroid was enlarged (weighing forty-six grammes): it showed numerous cysts, while many of the vesicles contained instead of colloid material numerous small cells; the interstitial connective tissue had undergone some fibrous increase.

The spinal cord presented the typical changes of tabes dorsalis. In the cervical region the degeneration was limited to Goll's columns. It could be traced up as far as the nucleus of the restiform bodies, but the restiform body, the solitary bundle, and other parts of the medulla were intact.

The sympathetic (right side only examined) was healthy.

The right vagus showed a few fibres in a condition of Wallerian atrophy. Various peripheral nerve trunks showed slight degenerative changes.

This is the first case combining the two conditions which has been fully examined *post-mortem*. Other cases have been reported clinically.

In this case the presence of locomotor is beyond dispute, while the examination of the thyroid, coupled with the clinical signs, also places the existence of exophthalmic goitre beyond cavil. For undoubtedly various symptoms may occur in locomotor resembling those of the other affection, such as permanent tachycardia, exophthalmos (due to paresis of ocular muscles), gastro-intestinal crises, and cutaneous anæsthesia; thyroid changes are quite unknown. Leaving out of question such cases of locomotor presenting these symptoms, there is no doubt that the spinal sclerosis and true exophthalmic goitre do co-exist. The problem is the connection between the two. The supposition has been made that the spinal degeneration has spread upwards and involved the medulla. This important case negatives such a supposition, and confirms the view that the cardio-vascular symptoms are merely by accident associated with the spinal change, as they may be associated with syringomyelia. The integrity of the restiform bodies is highly important, as opposing the theory advanced by Mendel, that those structures are the seat of the lesion in Graves' disease.

Another point of interest is the presence of tuberculosis. From our acquaintance with *post-mortem* records of Graves' disease we are disposed to say that tubercle is very rare. Greenfield raises the same question in contrasting Graves' disease and myxœdema (in which tubercle frequently appears). But we are not yet in a position to be dogmatic on this point.

Reymond[16] reports the case of a woman of 45, in whom acute symptoms of exophthalmic goitre came on very suddenly, owing to severe anxiety, and proved fatal in 14 days. The temperature ran up steadily during observation, reaching 104° on the third day. She had had continuous tremor for more than a year before she came under observation, so that this must, we think, be regarded as a "fruste" case terminating suddenly, rather than an acute case of very short duration. At the termination there was constant vomiting and dysphagia, even to liquids. *Post-mortem* mitral incompetency, which had manifested itself during life, was discovered.

The thyroid was enlarged, contained one cyst, and

microscopical examination showed hyperplasia of the epithelial cells, but none of the characteristic follicular structure. We presume this may be taken to correspond to the catarrhal hyperplasia described by Greenfield and others.

The thymus was present. This is by no means uncommon. Not only is the thymus persistent in many cases of Graves' disease, but in two instances it has been at least four inches long, and in Mr. Spencer's case it was larger than the head of the normal pancreas.

The peri-bronchial and tracheal lymphatic glands were also enlarged.

F. Müller[17] reports three cases of great rapidity and acuteness, all dying two or three months after the beginning of the illness; a fourth patient was discharged apparently dying in five months. He also reports a case of ten months' duration (sub-acute) which ended very rapidly. Several of these presented bulbar symptoms (as Reymond's case with dysphagia), irregular fever, and grave psychosis, such as restlessness, somnolence, delirium, and delusions of a terrible kind. Death occurred in all from cardiac failure.

All four cases presented *post-mortem* hyperplasia of the thyroid, and enlargement of the cervical lymphatic glands, with numerous minute hæmorrhages in the glands. Three showed small recent hæmorrhages in the medulla, chiefly in the rhomboid fossa, and near the vagus nuclei. One of them had considerable degeneration of the trunks of the vagi, the recurrent laryngeal, and cardiac nerves. In one only some slight œdema of the spinal cord and bulb could be detected, and no other nervous lesion.

Müller sums up recent theories on the subject thus:

(1) The sympathetic must be definitely thrown over on clinical, physiological and pathological grounds.

(2) The medullar theory, more usually accepted, he pronounces more rational, but insufficient to explain the whole clinical picture.

(3) That the cerebrum is affected is undoubted from the frequency of psychoses, convulsions, hemiplegia and hemianopsia.

(4) That the cord also suffers is proved by the frequency of loss of patellar reflex, and the occasional co-existence of tabes, syringomyelia and Duchenne-Aran atrophy.

He concludes that the morbific agent is not limited to the medulla, but affects all the nervous centres, including probably the

sympathetic. The variations and irregular progress of the disease militate against any "organic" lesion; the bulbar hæmorrhages are of no moment, being probably produced in the act of death. The inconclusive characters of his *post-mortem* are in favour of a purely "functional" disturbance of the nervous centres. Two factors in causation he considers very important: first, a neurotic predisposition either hereditary or acquired (by depression or shock); more important still, some thyroidal change. In support of the latter point he cites the now numerous cases of old-standing goitre preceding definite symptoms; the value of surgical interference: the obvious relation of myxœdema with exophthalmic goitre: the fact that the goitre is not merely hyper-vascular, but is always found to have undergone a definite hyperplasia of its tissue, and lastly, the very constant enlargement of the cervical lymphatic glands.

He deduces the supposition that the train of phenomena is the result of a functional disturbance of the thyroid, the altered secretion of which acts as a toxin, first to the lymphatic glands, and ultimately to the whole nervous system.

As evidence of our full acquiescence in these views, we may perhaps point out that an almost precisely similar summary formed the conclusion of our paper at the Medical Society in October (1893), when we had not had the advantage of seeing Professor Müller's article.

Bruhl,[18] in a long and verbose paper, loses himself in a forest of fallacies. He reports in detail the history of a woman, who died aged 61. She had at 39 developed a goitre, and came of a goitrous family, also highly neurotic, and from a goitrous district. Seventeen years after the appearance of the goitre exophthalmos was noticed, and she subsequently showed a rapid and irregular heart, and œdema of the legs. A careful *post-mortem* revealed a thyroid studded with cysts, and largely invaded with fibrous tissue, the heart hypertrophied, with some thickening of the aortic valves and the vessel itself. Nothing abnormal was discovered in the central nervous system save a small hæmorrhage in the fourth ventricle; no details are, however, given as to the histological methods employed in this examination. M. Bruhl will commit himself to no definite opinion, but is unwilling to accept this case as one of true exophthalmic goitre, and seeks to relegate it to a class of "false exophthalmic goitre," in which symptoms resembling the typical symptom-complex are presented by patients suffering from simple goitre. His grounds of argument are the district origin of his patient, her previous goitre,

her age, the pathological condition of the thyroid, and the organic condition of her heart. He is, however, doubtful whether to assign her cardiac disturbance to thyroid pressure or to muscular degeneration in the heart itself.

There is no question that in goitrous districts (*e.g.*, Canton Vaud, according to Rendu) exophthalmic goitre is unknown, but this may only mean that among a mountain peasant population such a neurosis is uncommon. There is no evidence to show that, given other hereditary neuroses, Graves' disease may not be found in the midst of endemic goitre, and the number of instances is now considerable in which old-standing goitre has been followed after many years by the signs of exophthalmic goitre.[19]

We have no reliable data as to how late in life exophthalmic goitre may appear, but Russell Reynolds remarks: "Although more than half of the cases (forty-eight in number) were under thirty, yet the influence of age is reduced to *nil* if regard be paid to the number of people living in the succeeding decades."[20]

Of thirteen cases of our own, carefully watched for some years, three presented their earliest symptoms from 40 to 50 years of age, and one at over 50.

His remarks on the pathological conditions of the thyroid will not bear the light of modern researches. Professor Greenfield and (we have just seen) M. Joffroy both show that cystic and fibroid changes are the rule in old-standing Graves' disease; and the notion that the goitre is usually "pulsatile" or "vascular," is entirely erroneous.

Bruhl insists that Basedow's disease may be well marked without goitre, and may even ensue after ablation of the thyroid.[21] It is, however, very easy to leave behind a fragment of thyroid or an accessory gland.

Our own reading of this instructive case is that it was a case of ordinary Graves' disease, occurring in a goitrous subject, of neurotic ancestry and personally neurotic tendency; that the preponderance of cardiac symptoms were due to the existence of ordinary cardiac and aortic degenerations, natural at such advanced age. Unfortunately, no note is made of the presence or absence of many ordinary signs, such as gastro-intestinal disturbances, though it must be remarked that no tremor was found present.

Rockwell[22] considers the therapeutics of the disease from a study of forty-five consecutive cases. Electricity is in his opinion the most valuable reagent. Convinced of the value of local gal-

vanisation, directed to the sympathetic and præcordium, he insists that it should be accompanied by general faradisation. This corroborates the views expressed by Dr. H. Lewis Jones ("Medical Electricity," p. 312) and Max Weiss.[23] Rockwell also insists that general galvanisation should be employed also, side by side with the other methods, which we find to be the opinion of Mr. Cardew;[24] but Rockwell would employ far stronger currents than have been used formerly. For application to the sympathetic and vagus, instead of two or three milliampères (Cardew) he has employed ten or fifteen, though it is rarely possible with ordinary electrodes, to exceed this limit. For greater strengths large electrodes of clay are to be applied to the nucha (cilio-spinal centre), and the solar plexus: with such terminals he has frequently used currents of *sixty* milliampères, finding great benefit. Such currents are, of course, only admissible in the hands of trained experts, and would require caution and judgment.

Professor Putnam[25] has produced several papers on this subject lately, dealing chiefly with the question of operation on the thyroid. He gives a tabular analysis of thirty-one collected cases of operation, mostly partial or complete thyroidectomy, and attacks the two questions of the value and the danger of operation. Freiburg,[26] of Cincinnati, has collected forty-one cases, and puts the mortality at six per cent., while undoubtedly a great improvement in results has accompanied the adoption of more perfect surgical methods in the last few years. But in spite of this improvement, and the comparative restoration to health which has often ensued, there are special dangers. Patients are liable not only to sudden death within a few days of operation, but they may suffer extreme prostration, high pulse rate, laryngeal paralysis, dyspnœa, vomiting, and pulmonary complications, even when the wound is quite satisfactory.

Paresis of the larynx is very common; any pre-existing paresis contra-indicates operation, and careful examination by a laryngologist should be made beforehand. Putnam thinks that goitrous patients do not always bear anæsthetics badly, though we find[27] that many Swiss surgeons have abandoned anæsthetics in large operations on the thyroid. There seems no evidence that the nature of the thyroidal enlargement has any bearing on the result, and the forms of change in the gland are numerous. Nearly all the cases have been those of large goitre with urgent symptoms, and we yet need information as to the effect which is produced by operation on small goitres in subjects with mild symptoms.

Putnam raises the question whether the dangerous symptoms are not due to rapid absorption from the divided gland of a large amount of thyroid secretion. This is by no means improbable, and the operation practised by M. Poncet (of Lyons) seems calculated to prevent this, as also to avoid injury to the recurrent laryngeal nerve.[28] This procedure to which the name "exothyropexia" is given, consists in shelling out the gland, through a median incision, with the fingers and blunt instruments, and leaving it covered by dressings, and attached by its deep connections on the surface of the neck, where it dries up and atrophies. This operation is less applicable to cystic goitre than to general enlargements.

In a monograph shortly to be issued,[29] of which we have been favoured with an advanced copy, Dr. Edward Blake reviews some of the evidence bearing on the etiology of myxœdema and the goitres. He assumes as established that exophthalmic goitre is a toxic neuritis of the medulla, extending at times to the pons and cervical sympathetic. He lays stress on the resemblance between the effects of this toxine, whatever it may be, and those produced from the absorption of pus; while he seeks to prove that a variety of conditions, such as rheumatism, exophthalmic goitre, endemic bronchocele, are all due to an organic poison akin to the nitrites in their action on the medullary vasomotor centres, and that in a large proportion of cases their toxines are derived from the slow absorption of pus, from carious teeth, or diseased genital organs (in the female). The brochure is suggestive, and the evidence is drawn from a wide range of literature, but it is ill-arranged and not fully digested. Dr. Blake fails, we think, to disprove that all cases of so-called Graves' disease show primarily some thyroidal enlargement, and that though the question is still *sub judice* the balance of evidence is in favour of a thyroidal origin for the nerve poison.

Conclusion.—It is obvious that all over Europe there is a gradual growth of the opinion that this symptom-complex is due to the production (or non-elimination) in the thyroid itself of some toxin, which acts on the whole nervous system, even to the periphery, though the brunt of its action falls on the vasomotor centres of the medulla, and some neighbouring centres. This position is adopted in Germany by Möbius, Wette and Müller, in France by Joffroy and Renaut, in England by Byrom Bramwell, George Murray and Greenfield. The question of the exact *rôle* played by the thyroid change is difficult; it is clear that all morbid changes in the thyroid are not operative to produce Graves'

disease, but all the cases (in which the gland has been properly examined) show two common factors—cell proliferation, and diminution of colloid. Even so we are in the dark as to whether this morbid change is primary or secondary to some vasomotor disturbance elsewhere, and also as to whether the alteration in thyroid secretion will produce the same effect in a healthy nervous system: probably some nervous systems are more susceptible than others to the toxic influence, just as they are to alcohol and lead; in fact the susceptibility of young women to plumbism and Graves' disease is strikingly similar.

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HYSTERIA AND NEURASTHENIA.

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(*Concluded from page* 178, *Spring Number.*)

IV.—Papers on Hysterical Disorders of the Organs of Vision.

PROFESSOR PICK[35] thinks that the exact methods of diagnosis in use in ophthalmology are just suited to solve the difficult problem, which has been called attention to by Charcot and Buzzard, of the distinction of hysterical symptoms from those of organic nervous disease. He therefore reports two observations of the kind which also show the unmistakable influence of hypnotic suggestion.

The first patient was a girl of 24, who during infancy suffered from left ptosis and inability to raise the eyelid. Vision during childhood was so defective that she could not learn to read or write, and there was loss of movement of the eyes upwards. Except for the defective vision her health and intelligence were good, and she was employed in housework. Two years before she came under the author's notice she had been treated at an eye hospital, and the diagnosis made of congenital amblyopia. V.=$\frac{2}{60}$, similar in both eyes, with nystagmus; the fundus was normal; except for ptosis and nystagmus the eyes could be freely moved. At this time she suffered from convulsive attacks and anæsthesia of undoubtedly hysterical character, the attacks being followed by hysterical delirium. After this the amblyopia was increased to complete amaurosis. The patient was a small, strongly-built, and well-nourished woman. The skull showed great abnormality in form. The teeth were irregular and bad, the left ear deformed. The eyelids were kept closed, and could only be

opened by a great effort, which was accompanied by associated action of the facial muscles. When the patient sat up her head was bent backwards, and an order to bend it forwards only resulted in its being bent still further backwards; passive movement met with strong resistance. Full extension of the upper extremities at any joint could not be accomplished voluntarily or passively. The legs were extended, and in walking the feet were dragged along the ground; she walked with extended legs and short steps. There was anæsthesia over the left side of the body and on the right leg and the back of the right hand. The patient answered all questions readily and intelligently. After an attempt at producing transference by means of a magnet the loss of sensation was transferred in the *upper* part of the body from the left to the right side, and the eyelids could be raised voluntarily for 6-8 m. without producing over action in associated muscles. Repeated applications of a magnet with one pole over each eye resulted in a remarkable improvement of vision, and two months after admission she could count fingers at 210 cm. without glasses, and at 3 metres with —3D. Later she improved still further, and could read the last row of Snellen's (capitals) types. The eye movements were fully co-ordinated outwards, upwards, and downwards, and convergence was promptly effected in a good light. The contractions in the hands grew less, and the convulsive attacks ceased. In addition to amblyopia and disturbances of motion of the eyes of congenital origin, on whose pathogenesis one cannot enter further, but which, in the author's opinion, are to be considered as related to the abnormal form of the skull, there developed on the one hand amaurosis, on the other external ophthalmoplegia of hysterical character. The proof that the last symptoms were hysterical is afforded by the fact that they came on after a convulsive attack amongst other hysterical symptoms; the ophthalmoplegia showed these characters lately regarded as hysterical; and under the influence of suggestion both returned to the state in which they were before the onset of the hysterical symptoms.

On one occasion the placing before the eyes of a —3D. lens produced improvement of vision, but a similar effect was also given by a strong + glass, and also by a tinted plane glass whose power of refraction was *nil*.

The author attributes the amaurosis to the influence of suggestion, in the hysterical condition of the patient the presence of disturbances of the oculo-motor muscles being sufficient to determine the incidence of the hysterical affection in the eye.

The second case was one in which, at the autopsy, there were found areas of cerebral softening, following thrombosis of the basilar, right and left posterior, inferior cerebellar arteries, and "art. proff. cerebri," suppurative salpingitis and chronic adhesive perimetritis. There was also consolidation of the lower lobe of the left lung. During life the patient suffered, amongst other nervous symptoms, from loss of sight, and from ext. ophthalmoplegia. Six months before death, in the early stage of these disturbances of vision, she was treated by Dr. Bayes by suggestion, by means of which vision within a few days was improved (from $\frac{4}{60}$ to $\frac{1}{36}$); the patient, however, left off the treatment because she had not improved enough to be able to read.

Prof. Knies,[36] in considering the question of unilateral visual disorders, says that the occurrence of a one-sided disturbance of vision is denied by some, affirmed by others. The question turns on the point, whether a lesion of the optic paths seated centrally to the chiasma, optic tract and primary optic ganglia can produce disturbance of vision on one side? Clinical experience shows that lesions of the optic radiations, or part of the cortex concerned in vision, produce disturbances of vision, more or less homonymous in character, on both sides. In those cases in which a one-sided visual affection has a central cause, visible anatomical alterations are not present, although for other reasons a centrally seated cause is to be assumed. A type of one-sided central disturbance of vision is that so frequently met with in hysteria, for that unilateral visual disorder is met with, and not infrequently in hysteria, cannot be doubted on account of the many observations of competent observers; and it is impossible to believe that it really exists on both sides, one having been overlooked. [It is now generally accepted that although the affection of vision predominates on one side in hysteria, careful examination nearly always detects some defect in the other field, although it may be slight.] The author is of opinion that in the majority of cases of hysteria the visual disturbance is unilateral, and suggests that when it is bilateral the central disturbance may be so too. The chief features of the affection are: (1) It is not hemiopic in character. (2) Failure of sight occurs in all grades, but seldom goes on to blindness. (3) Reaction of the pupil to light is not affected in mild cases, in complete blindness it is generally lost, but may be preserved. (4) Concentric contraction of the visual fields is found, and is in nowise always proportional to the degree of disturbance of vision. (5) The failure of colour vision is generally a typical one, and corresponds to the difference between the

perception of colours in a strong light by the periphery of the retina and fovea centralis. In general, the disturbance of colour vision corresponds to the degree of narrowing of the field, and to the deficiency of central acuteness of vision.

That these visual disturbances are of central origin is shown by their occurring in connection with other hysterical symptoms of undoubtedly central origin; they are susceptible to psychical influences; they quickly come and go, and are often amenable to suggestion; they may disappear or be transferred to the opposite side, by the application of plates of metal; they may be removed by altogether insufficient means, such as putting a plane glass before the eyes; and in spite of the great contraction of the visual fields, the patients have no difficulty in seeing their way about; these last conditions make the resemblance to malingering very close. The fundus oculi is normal, and no changes appear in it after these visual troubles have lasted for years. Although certain experiments on animals (Lannegrace) appear to indicate the presence of a unilateral crossed visual disturbance of cerebral origin, yet in face of the very different formation of the central visual apparatus in animals and men, caution must be exercised in arguing from one to the other. The most serious objection to the conception of a one-sided visual disturbance of cerebral origin is that there is no anatomical evidence of a second crossing of the optic tracts above the primary optic ganglia (ant. corp. quad. and corp. genic), which on this supposition would be necessary. The author thinks that the chief facts point to a position distal to the chiasma as the seat of the lesion, and that the optic nerves may be more or less compressed at the optic foramen, and so their power of conduction impaired, but not destroyed. Thus the peripheral parts of the retina, supplied by fibres which lie in the periphery of the nerve at the optic foramen, are the most affected. The nutrition of the retina does not suffer because its vessels are not affected. In the sclerosed areas of multiple sclerosis nerve-fibres are often present, which are still capable of conducting impulses, but have lost their medullary sheaths. The most likely form of compression at the optic foramen is an annular one; this might be produced by vascular dilatation at this position, and compression of the nerves not passing on to destruction of them result. This opinion is supported by one actual observation. Leber[97] has anatomically proved, in a long-standing case of hysterical visual disturbance, the presence of atrophied fibres on the periphery of the optic nerve, especially at the position of the optic foramen. He thinks,

however, that such trifling anatomical changes cannot be the cause of the visual troubles. Prof. Knies admits that the retention of the light reflex, often present in complete amaurosis, militates against his theory, but thinks that this difficulty may be got over by supposing that afferent impulses can pass to the lower centres, but that the obstruction so weakens them that they cannot reach the cortex.

To explain the frequent dependence of hysterical visual disturbance on cerebral causes, the nature of the process must be vasomotor. If this cerebral vasomotor disturbance is unilateral, defects of sensation and motion on one side will also appear. It is surely no mere accident that the only nerves which are hardly ever affected with hysterical paralysis are those which do not pass through narrow canals, namely, the nerves which pass out through the sphenoidal fissure. (The existence of a form of ophthalmoplegia externa in hysteria is now beyond question; see Dr. Bristowe, "Lectures and Essays on Diseases of the Nervous System.")

The "central" character of hysterical symptoms is accounted for by the grouping of nerve fibres in the intra-cranial canals in the same order that they occupy within the brain itself.

On these grounds the author concludes that hysteria is essentially a cerebral (and spinal?) disturbance of innervation, a great part of the symptoms arising from lesions of nerves and nerve roots, which pass through narrow canals. The common exciting causes (psychical disturbances) of hysteria produce a high degree of contraction of the (cerebral?) vessels, followed by a corresponding secondary dilatation. In those cases in which there is no exciting psychical cause, there is a manifest hysterical disposition, frequently hereditary. This may possibly be due to a deficiency of the medullary sheaths of the nerve fibres, or to defective development of them. This is pure hypothesis, but the author thinks that a quantitative estimation of the medulla of nerve fibres and of the condition of the intervertebral canals might lead to some important results.

As the result of the examination of over sixty nervous individuals, Dr. Wilbrand[38] finds that a symptom complex is present in all forms of the traumatic neurosis, consisting of contraction of the visual field, loss of acuteness of central vision, weakness of accommodation, and a great number of other visual troubles. Seeing the frequency of narrowing of the visual field and of asthenopia in all forms of nervous conditions, it would be remarkable if they were found in the traumatic neurosis so seldom

as some authors say. Dr. Wilbrand, from his own observations, is of opinion that contraction of the visual field is of very frequent occurrence, and attributes the statements to the contrary of other observers to defective observation.

It may escape notice, because although an objective symptom, complaints may not be made of asthenopia, or the contraction may be present in one eye only, and that one not be examined. He attaches as much diagnostic value to slight as to great contractions of the fields. The question of simulation he thinks can be easily settled, by reason of the many methods of perimetric investigation at our disposal by which simulation can be readily detected.

Drs. Franki Hochwart, and Topolanski,[20] from observations in Nothnagel's clinic on non-traumatic hysteria and in neurasthenia, give the following results. In hysteria without sensory disturbance, there is generally no concentric contraction of the visual field, nor sensible disturbance of colour and light perception (fourteen cases). In hysteria, with sensory troubles, perception of light and colour may be quite normal (eleven cases). Yet although the field of vision and perception of light may be normal, the power of recognising one or the other colour may be somewhat disturbed in cases of hysteria, whether sensory disturbances are present or not.

The concentric contraction of the visual field is a pretty frequent symptom in hysteria with sensory affection (ten cases), this contraction is as a rule most pronounced on the anæsthetic, but is generally present on the other side too. With the contraction of the field of vision there is often considerable disturbance of the perception of light and colour, which in many cases corresponds in degree to the contraction of the field. The size of the field and together with it the light and colour sense fluctuate with changes in sensibility.

As an isolated symptom the authors have also observed an increase in size of the visual field and true hyperæsthesia for light and colours.

In neurasthenia there is generally no concentric contraction of the visual field (thirty-one cases). Perception of light and acuteness of vision are normal. Only occasionally is there a certain amount of loss of colour-perception, generally for blue.

The conditions in traumatic hysteria and in neurasthenia were the same as in non-traumatic cases. Over one hundred cases were examined. The authors emphasise the fact that from the method of examination there could be no question of simula-

tion. If suspicion of simulation be aroused in the investigation of the field of vision, the testing of the perception of colour and light can be used as a confirmatory test. Decided agreement in the results of all methods of investigation shows the correctness of the observation. Where there is no contraction of the field of vision in hysteria, loss of colour-perception may be present, and together with the recognition of the sensory disturbance may sometimes aid in the diagnosis. In hysteria without objective symptoms as well as in neurasthenia it may perhaps be the only symptom capable of demonstration.

Drs. Mitchell and de Schweinitz[40] have studied eight cases, presenting various degrees of hysterical hemianæsthesia and alterations in colour vision.

"It is evident from an examination of these cases that in no instance was there loss of the colour-sense, the achromatopsia of Galezowski, nor was there any disappearance of the colours in a constant order. In the two cases of complete anæsthesia there was no departure from the normal sequence of the colours; and, practically, no limitation in the area of the field of the one (Case I.), and but slight restriction in the other (Case II.).

"In the cases of partial anæsthesia, in one (Case VI.) no abnormality for blue and red existed; green was by contrast contracted, and in two others (Cases III. and IV.) there was concentric irregular contraction of the colour-fields; but in all three the normal sequence of the colours was undisturbed.

"In two cases (IV. and V.), while the colours were correctly appreciated, their natural order was changed, the blue, red, and yellow lines crossing each other in such a manner that the red field became in some meridians the greatest in extent.

"In only one case (Case VIII.) was there an approach to complete transposition of the colours so that the red became the largest field, followed by yellow, green, and blue. In none of the cases was marked amblyopia present, except in Case VII.; here the deficiency in visual acuity was due to a high grade of hypermetropic astigmatism and concomitant convergent squint.

"We have had the opportunity of studying two cases of absolute hysterical hemianæsthesia, presenting the phenomena of crossed amblyopia. In one, a woman aged 25 years, seen by the courtesy of Dr. H. C. Wood, these symptoms appeared several months after the shock of a railroad accident. There was complete right-sided hemianæsthesia and loss of the special senses on the anæsthetic side, with amblyopia in so high a degree that quantitative light-perception was absent. The vision

of the left eye was reduced to one-ninth of normal; red was the only colour correctly appreciated, and there was left lateral hemianopsia.

"In the other case, examined by the kindness of Dr. G. Oram Ring, who will publish the notes in detail, precisely the same conditions obtained, except that the amblyopia was less marked upon the anæsthetic side. In both of these cases the ophthalmoscopic appearances were normal.

"If, as may fairly be supposed, these eight cases, taken at random from a large service, represent the ordinary conditions, then our conclusions as to the occurrence of achromatopsia with hemianæsthesia must be very different from those of the European observers. The patients were selected for study because they all presented greater or less degrees of loss or change of sensibility. As to this, it should be said that we cannot find any statement by the French writers, who are the original students of this subject, concerning the relative frequency of hemianæsthesia in all hysterical cases, except statements like that of Dr. Bernutz, already quoted, that it is 'one of the primordial symptoms,' or Charcot's remark in exactly the same sense, which would imply that it was present as an almost constant characteristic. Further, as to generalised anæsthesia, or, more properly, analgesia, Bernutz calls it 'infinitely rare,' and Charcot 'relatively exceptional.' The results of the ward-service as well as of the out-patient department of the Infirmary for Nervous Diseases would not allow us to make assertions which should corroborate these. These services may, we think, be considered to represent well at least the Eastern United States, as patients come from all of them to seek treatment, and our eight quoted cases are natives of several States—Pennsylvania, Wisconsin, New York, Virginia, and New Jersey. Every case differs in the *quality*, too, of the sense-changes, and some have that very curious symptom of ability to feel heat, when great, as pain, while they cannot appreciate a needle-thrust."

In a further paper by the same authors they say that in seven cases in which one or other of the varieties of anæsthesia was a marked symptom, the most usual change was a more or less complete transposition of the red and blue lines, the red being the greater in extent. In several there was also great contraction of the visual field. In one case only in which the anæsthesia was of minor character, were the form and colour fields normal in extent. In the patient who exhibited the least marked general hysterical symptoms, there was the most remarkable deviation from the

normal standard of the visual field. In cases of hysterical hyperæsthesia the disturbances in the visual field are of secondary importance; the deviations from the normal being so slight and uncertain that they could not be regarded as peculiar to that condition.

In cases classified as major hysteria there was no reversal (of red and blue fields) and no anæsthesia (in two there was contraction of the visual field), whilst in the "local" hysterias, although there was no anæsthesia, there was more or less complete transposition of the red and blue lines.

In cases classified as neurasthenia, the changes in the visual fields were not very marked, with one exception, in which the fields of vision were characteristically hysterical in composition.

In the others, if they exhibited any change it was that one most often seen in anæsthesia of the retina as described by Wilbrand. In a case of spinal injury there were characteristic (hysterical) changes in the normal appreciation of colours. The authors conclude:—

(1) Achromatopsia is not present in the American cases.

(2) Reversal of the normal sequence of the colours so that the field for red is the largest usually accompanies anæsthesia, but disturbance of colour sense is not necessarily associated with anæsthesia.

(3) The field for green is relatively more, and more often contracted than the others.

(4) Colour sense alterations are more common in hysteria than in neurasthenia, but though of diagnostic importance this is not a certain distinction, as they may be absent in typical hysteria and present in neurasthenia.

(5) One case seemed to point to the possibility of an abnormally acute colour sense in the rare cases of one-sided or general hysterical hyperæsthesia.

(6) The disturbance of colour sense is not proportionate to the severity of the hysterical manifestations.

(7) The most frequent changes in the visual fields in hysteria are:—

(*a*) Simple contraction of fields for colour but not for form.

(*b*) Contraction of fields for both: the field for green the most.

(*c*) Partial or complete reversal of normal sequence in size of colour fields, the red being most commonly the largest. The colour fields may be normal in extent or contracted.

(*d*) Loss of parts of the visual field, *e.g.*, in form of hemianopsia, or paramount contraction of the field on one side, generally the anæsthestic side of the body.

V.—Papers on Hypnotism, Hysterical Somnambulism and Double Consciousness.

Dr. Azam[42] has collected in his book a number of papers written by him on these subjects. The works of Braid drew his attention to the study of hypnotism, and he reprints here a paper on hypnotism published in 1860, and of historical interest as the first publication in France on the subject; in this paper he gives an account of his own experiments on the lines already indicated by Braid. It is difficult to realise to-day, now that hypnotism has been the subject of such wide-spread research, that it required considerable courage in 1860 to undertake such studies, as they were then only likely to bring discredit and ridicule on the observer.

The second part of the book deals with double consciousness and somnambulism, and is largely made up of an exhaustive account of the celebrated case of Félida X., which was first brought to the notice of the scientific world in 1876. A short abstract of this well known case will be found in Professor James's "Principles of Psychology," vol. i., pp. 380 and 384. This patient passed into a "second" state at the age of fourteen, characterized by a complete change of character. In her "normal" or "first" state her memory only extended to the periods of her life passed in the "first" state, whilst in the "secondary" state, which in later life predominated to the almost entire exclusion of the first, she remembered the events of both "first" and "second" states.

Professor James thinks, on good grounds as it seems to us after carefully reading the case, that the secondary state was really the normal, and the primary the abnormal condition in this patient.

In a paper written in 1890, Dr. Azam says that the explanation of the curious fact that some persons have two simultaneous or alternating existences is to be found in an analysis of the phenomena of sleep, and especially of somnambulism, of which forgetfulness on waking is the principal characteristic. In this state the physical and mental activities are carried on in various degree in different cases.

The third part consists of memoirs on different subjects, being chiefly taken up by an elaborate and interesting study of the nature and origin of character, both in health and in disease, in individuals and in nations, and under different conditions of the environment.

Hypnotism.—Dr. Löwenfeld writes that hypnotism in hysteria and neurasthenia acts in several ways.

(1) Production of the hypnotic state only, a state akin to natural sleep, is useful in irritable conditions of the nervous system, in insomnia, certain forms of headache, mental anxiety and perturbation.

(2) Suggestion: Very often a condition of deep sleep is not necessary for suggestions to take effect. There are also persons so amenable to suggestion that hypnosis is unnecessary; this is especially the case in children. In other persons suggestion can only be effectual during deep sleep. Suggestions must be adapted to the condition of the patient, to his mental individuality, and to the special nature of the symptoms to be removed.

(3) Method of Breuer and Freud, see p. 125.

Therapeutically suggestion is of value in enabling isolated disturbances originating in the organ of mind, the cerebral cortex, to be acted upon apart from the symptoms of disease which may be associated with them.

Hypnotism can by no means remove the general hysterical or neurasthenical condition, but can only act on special symptoms.

As to the indications for hypnotic treatment there is much difference of opinion: many would keep it as a last resource, others would employ it at once whenever a suitable case presents itself. The author prefers the use of other measures when they will give similar results to hypnosis, and generally does not make use of hypnotism in such cases on the ground that a psychical influence (non-hypnotic suggestion) on the patient may be associated with every kind of treatment—in a certain sense *is* always so associated—and that hypnotism will not effect a cure more promptly or decidedly.

Specially capable of influence through hypnosis are hysterical symptoms of the more fixed character, paralysis, anæsthesia, hyperæsthesia, contractures, and frequently recurring convulsions, and many neurasthenic symptoms, such as loss of sleep, of appetite, and uncontrollable ideas. The drawbacks to hypnosis are the uncertainty of its action, not every patient being hypnotisable; that in full hypnosis with pronounced suggestibility, therapeutic suggestions may have no effect; that the results are often temporary, patients are relieved immediately after hypnotism, but by the evening or the next morning all their troubles have returned; and that one only out of many symptoms may be influenced by it. Hypnotism is in general more useful in hysteria than in neurasthenia. The failure of suggestion is partly attributable to the fact

that to be effectual its influence must last into the post-hypnotic state, into the time after hypnosis, and this demands a high degree of susceptibility to suggestion.

The author's experience leads him to refer the unfavourable effects of hypnotism largely to want of adaptive skill on the part of the hypnotiser. In the neurotic who are prejudicially affected by procedures quite harmless in the healthy, this is a fact of especial importance, and the use of hypnosis demands prudence and careful adaptation to the patient's individuality. In persons subject to hysterical attacks these may be brought on by hypnotic procedures. Just as other therapeutic measures, cold baths, massage, &c., may have bad effects on some individuals, so may hypnotism, but this mode of treatment prudently carried out by an expert, does not certainly entail more risk in itself than most of the other means of cure daily employed in nervous diseases.

Hypnotic treatment should not however be undertaken too lightly. The possession of a battery does not *ipso facto* constitute a skilled electrician, and the mere knowledge of certain methods of inducing hypnotism does not entitle anybody to put them into practice.

The full description by Charcot of the "greater hypnotism" with its distinct phases of cataleptic, lethargic and somnambulistic conditions, has not been confirmed nor observed outside the Salpêtrière by the many investigators of hypnotic phenomena. That the observers at the Salpêtrière saw what they described Dr. Löwenfeld does not doubt, but he thinks that the phenomena were excited in persons prepared for them, and easily influenced. He considers that further experience has shown that the hypnotic state is not confined to persons of hysterical dispositions.

Dr. E. Mesnet[45] reports the case of a girl aged 17 who had suffered from hip disease, and had always been delicate and anæmic, but was never troubled with nervous disorders until March, 1872, when she had rachialgia with loss of power and sensation in the legs. From being morose and uncommunicative she abruptly underwent a change in character, becoming talkative and gay. In April, after breakfast one day, she suddenly lost consciousness and fell into a state of sleep, which lasted for an hour and from which she could not be roused by any means. When she awoke she expressed great surprise and had no memory of what had passed. The attack returned regularly for four days at the same hour, and on the fifth a second one of the same kind came on after dinner. There was complete relaxation of the limbs, loss of all the senses for communication with the

outside world; the eyes were closed, with flickering of the lids. When she awoke she complained of acute pains in the head and stomach, implored aid from her relatives, but could neither hear their replies nor see them, the eyelids being closed. She attempted to escape out of the room first by the door then by the window. She struggled violently with those who opposed her movements, and showed an extraordinary amount of strength, requiring three or four persons to hold her. She afterwards talked rapidly in a delirious way for a time, then fell into a sleep like that of the beginning of the attack, and after five minutes awoke, rubbed her eyes, asked what had passed, and was surprised at the disordered state of her room, remembering nothing of the attack. The next day she had two attacks at the same hours; after twenty minutes' lethargic sleep, a period of agitation with violent declamation followed, then succeeded all sorts of violent movements, of gymnastic exercises, in which she overturned everything within reach and struggled violently when resisted, then came a period of sleep of five minutes' duration, after which she awoke, unconscious of what had passed. These attacks recurred twice a day at the same hour, varying only in duration and in intensity and in the effect of impressions received during wakefulness on the phenomena of the somnambulistic states.

All kinds of treatment including hypnotism were tried without avail. By June she had become much worse, the attacks coming at the same time but being so prolonged that she was only in her normal state for fifteen hours out of the twenty-four.

On July 1 she was removed to the Hospital Saint Antoine, and there impressed with the fact that she would be cured. As a matter of fact, without any other treatment the attacks ceased on July 3, and she returned home well on the 14th.

Dr. Cullerre[41] gives an account of a similar case of hysterical somnambulism in a girl of 20. The patient was subject to attacks of somnambulism, which would come upon her at any time, but almost always at night; in them she several times walked about the village in which she lived half naked. She was of healthy appearance; there was general cutaneous analgesia, with complete hemianæsthesia on the left side; on which also marked muscular weakness, diminution of visual acuity and dyschromatopsia existed. Whilst under Dr. Cullerre's care she had twenty-eight nocturnal attacks of somnambulism, coming on at variable hours, and varying in duration and intensity from half an hour to two hours of simple delirium, the patient not leaving

her bed, to violent scenes of true somnambulism with uncontrollable actions lasting several hours. The events of the day always had a great effect on the return and character of these attacks—the most violent following lively emotions, especially disagreeable ones.

After some months she passed into a very excited mental state, one of exuberant gaiety (expansive). She recovered after some months, but relapsed after a fright a month later and was re-admitted to the asylum. She suffered then from analgesia extending nearly over the whole body. In two months the somnambulistic attacks ceased, but her character changed; from being calm and reasonable she became mischievous, with accesses of maniacal exaltation; after a few weeks she had an hystero-epileptic attack, after which her disposition improved again and at the same time the fits of somnambulism returned. The patient could be hypnotised by direct means, and the hypnotic state was that of somnambulism with closed eyes; but the occlusion of the eyes was not perfect, as she was able to recognise correctly objects such as photographs of persons known to her. There was anæsthesia except of hearing, which was abnormally acute. Contrary to the rule there was not mental passiveness. She would not, as a rule, accept suggestions made to her. Left to herself, the hypnotised patient became delirious, the delirium being a replica of that observed in the attacks of natural somnambulism. The only difference between the states of provoked and spontaneous somnambulism was that in the latter the eyes sometimes remained open. Attempts to suppress the spontaneous attacks by means of suggestion failed. In this patient it was not simply a state of noctambulism, but one of somnambulistic delirium which had always the same course and characters, in part composed of fixed hallucinations always the same, in part of varying ones, changing with the nature of the daily events in her life; thus this somnambulistic state presented the same features as those of the delirium of the third period (période des attitudes passionelles) of the fully developed hysterical attack, as described by M. P. Richet, except in one respect, the complete absence of memory by the patient of the somnambulistic attack.

Attacks of Sleep (Dr. Löwenfeld).—The absence of reaction during the attack of sleep to the different kinds of sensory stimuli, including painful stimuli, does not indicate that consciousness is abolished. Several observers have noted cases in which there was loss of reaction to external impressions—so-called "Scheintodes"—yet the sense of hearing was preserved. Hence the patient may

retain some memory of the occurrences during lethargy, but this memory may be very dim for the events of the attack, or fail altogether during a certain part of it. In the majority of patients no remembrance of the experiences during the attack of sleep remains on waking. During these attacks, which for the patients form a gap in their psychic life, a certain amount of mental activity is retained. As in natural sleep they change their position from lying on the back to the side, and may carry out movements of the head and arms. Thus one patient—who suffered often from chilliness—wrapped herself up on these occasions in a woollen covering, and if this was drawn away so that the upper part of the trunk and the arms were exposed, she wrapped the covering around her again. Another patient reacted to a suggestion made in her hearing whilst she was in lethargy that she should go to a concert if she awaked half an hour before the appointed time for it.

Hysterical Somnambulism.—Dr. Löwenfeld adopts Joseph Frank's division into (1) somniatio, (2) noctambulism. Several groups or varieties exist. Attacks of somnambulism may be attended by pronounced and easily recognisable mental disturbance or by such slight mental change that it can only be recognised by those who have an intimate acquaintance with the patient's normal state. As a rule the patient in his normal mental condition is unaware of all that has occurred during the period of hysterical somnambulism; only very seldom does a dim remembrance of single events of the somnambulistic state remain. The somnambulistic bears the same relation to the normal mental state that a tumour does to the rest of the body. The mental attainments and character of the patient in the two states are often different. The second or somnambulistic state may attain such a duration and importance that a doubling of the personality can properly be spoken of, a division of the mental life into two psychical existences distinct from each other.

In the majority of cases the patients are not in the full possession of their natural intellectual powers during the somnambulistic state. Prolonged somnambulistic conditions often follow convulsive seizures or attacks of sleep, and the return to the normal may be similarly brought about. Hysterical somnambulism differs from attacks of psychical epilepsy with ambulatory automatism in that in the latter no remembrance of the events that have taken place during them can ever be aroused, whereas in hysterical automatism, a return of the secondary state, which may be brought about by hypnotism, brings back

with it the remembrance of past acts of the original or of other previous attacks.

In concluding a paper on "Cases of Hysteria treated by Hypnotism," Dr. S. G. Webber,[45] of Boston, remarks: "The first is a complicated case. There was a peculiar condition of the nervous system, a condition of neurasthenia, also a hysterical state. In some way, which I do not understand, the patient came into a state in which she lost her memory of recent events, and her relation to those about her were changed to such an extent that she did not recognise those whom she had seen and known only a few hours previously. Then she returned to her normal condition, and had no recollection of what had just occurred. Her brain then became a dual organ; in some way the awaking of one set of ideas and associations in one group of nerve cells seemed to inhibit the activity of the group of cells which was cognisant of the other set of ideas and associations.

"Later, when I had put her to sleep, there was the manifestation of other phenomena; there was not only the two states of brain activity, but also the power or ability of receiving suggestions when all the mental faculties seemed to be dormant, and it would have been supposed that she could not hear nor the brain receive any impression, at least so as to have a permanent effect.

"Memory, consciousness and will were perverted, or in the artificial sleep they seemed to be abolished; certain groups of nerve-cells, which are usually active during waking hours, were inhibited in their action. For a while they were placed in an enforced quiet. This was seen in the second case. I could easily put the patient to sleep; there was no cataleptic state of the muscles; she simply went to sleep, a quiet sleep such as anyone might have, with dreams or without as the case might be. She was, however, insensible to all noises, was not disturbed by the rattling of paper, opening of table drawers, the slamming of doors outside, nor by my moving about the room. As soon, however, as I spoke to her, she showed that my voice reached her brain, there was a change in the expression of her face, and if I continued to speak she soon waked up.

"It will be interesting to compare these peculiar mental conditions with what is seen in an ordinary case of delirium, in which there is a more or less complete unconsciousness of one's surroundings. I will report a very simple case which resembles hypnotism.

"Miss A. has had severe headaches, especially at the time of her catamenia, and in these is sometimes delirious.

"March 20, was called to her about seven in the evening, as she was delirious. I found her lying on her right side; the muscles of her neck and arms would occasionally twitch, and she carried her hand to her neck every now and then, and showed by slight groans and sighs that she had severe pain. She seemed to imagine herself at home, and called her mother in a rather low tone of voice. When the nurse came she told her to go for her mother. She seemed to think I was her uncle Edward. She tried to get up once or twice, and said she must go or be going. There was no violence and no loud outcry; she was simply in a quiet delirium. She did not realise where she was nor what she said, yet seemed to realise that she was in bed, and tried not to uncover herself. Under the use of remedies and massage to the head she fell asleep, awoke at a slight noise, then went to sleep again, and soon woke up in her right mind, astonished to find me by her, and asked if she had done anything out of the way.

"In this case there was an entire misapprehension of her condition and surroundings. She was as much out of touch with those about her as the hypnotic, more so in fact. Yet she retained a certain amount of receptivity; she knew that someone was near, and made known her request that her mother should come. In this patient the memory and consciousness were perverted or suspended, the will was still active, but acted in an abnormal way, because it had not correct data to guide it. Ideation was more affected. In hypnotism there was a more orderly sequence of ideas; in delirium the sequence of ideas was less regular; slighter influences served to give rise to new ideas not in harmony with those preceding. It seems to me this may be one of the differences between hypnotism and delirium; not only are memory, consciousness and will affected, ideation is irregular and dependent upon accidental influences, arising outside or perhaps within the patient. In hypnotism, when ideation is affected, it is perverted according to the will or suggestion of another.

"It would be interesting to compare these two states with dreaming. This seems to be a modification of the above conditions, the cause being within the brain itself, or often coming from without. But in dreaming there is less order; the control of the thoughts, which is not wholly lost in hypnotism, and which still is found to some extent in delirium, is very imperfect in dreaming; the logical faculty is more affected, or it may be said the memory is more at fault and does not act to regulate the sequence of ideas, so these run riot in the brain, and the different

groups of nerve-cells are awakened to activity and silenced in a most unorderly manner, giving rise to the most absurd and incongruous grouping of ideas and images.

"Dreaming is likely then to be the least orderly of these abnormal activities. There are, to be sure, dreams which seem to be perfectly orderly and logical. Perhaps they are so, or it may be that they seem to have this order because the waking mind unconsciously reduces the mass of ideas to something approaching order. It would be interesting to follow the analogies in these states much further."

VI.—Papers on the Association of Hysteria with Organic Nervous Disease.

Dr. Babinski,[46] in discussing the association of hysteria with organic and other nervous diseases, says that the affections which are first and foremost, disseminated sclerosis, then tabes dorsalis, have been especially noticed as frequently associated with hysteria syringomyelia, Morvan's disease, Pott's disease, facial paralysis, the primary myopathies, neurasthenia, agoraphobia, onomatomania, chorea, and exophthalmic goitre. It is reasonable to suppose that if a slight injury or simple emotion can give rise to the appearance of hysteria, organic affections of the nervous system, with the profound changes that they entail, should have analogous effects. Such a combination of hysteria and organic disease is obviously likely to be a fruitful source of mistakes in diagnosis, and it is to failure of recognition of this combination that the opinion put forward by some persons that psychotherapeutics may modify or even cure organic disease is in part due.

Case 1.—A woman of 45, alcoholic, suffering from right hemiplegia, the arm being most affected, with rigidity of muscles and exaggeration of reflexes, as the sequel of an apoplectic attack six months previously. There was also right hemianæsthesia, with contraction of the right field of vision of the same duration. The sensory symptoms were removed by the application of a magnet.

Case 2.—A man aged 31, who had suffered from syphilis ten years before. The illness was of one year's duration, and consisted of progressive mental enfeeblement and loss of motor power. The mental state was one of complete apathy, with inaptitude for any intellectual operations. There was also a peculiar difficulty

in speech, trembling of the upper lip, and slowness of reaction of the pupil to light. The upper extremities were normal, the lower could be moved freely when the patient was lying down, but he could not stand or walk without support; when he tried to do so his legs gave way under him. Sensation was deficient over the legs; the tendon reflexes were normal. Micturition and defæcation were naturally performed.

Faradisation of the lower extremities, together with a statement made to the patient that electricity would cure his paralysis, resulted in immediate improvement, and in ten days' time he could stand and walk fairly well.

The author says that this case is one of meningo-encephalitis with hysterical astasia, but he admits that the cure was not complete, and that, therefore, the paresis of the legs might be in part due to the cerebral lesion, but he considers that it was principally due to a purely dynamic or hysterical disturbance associated with it.

Case 3.—A female cook, aged 42, suffered from hysterical fits, followed by choreiform movements which lasted for a year; this occurred when she was 21 years of age. In 1892 she was knocked down by a carriage and sustained a fracture of the right petrous bone with permanent facial paralysis; two weeks later, attacks of vertigo and loss of the power of maintaining her equilibrium prevented her from standing or walking. When lying down she had no vertigo and no loss of motor power except a little feebleness in the right hand, but when sitting or standing she swayed from side to side, and the gait in walking resembled that of a drunken man. She had also hemianæsthesia, together with dulled hearing and contraction of the visual field on the right side. These motor disturbances were cured very speedily by suggestion.

Case 4.—A peripheral neuritis of mild intensity affecting a woman who had formerly had hysterical attacks. The neuritis affected chiefly the sensory fibres, and its chief signs were lightning pains, absence of tendon reflexes, and diplopia. In addition were present symptoms, which appeared to be almost exclusively due to hysteria; these were muscular enfeeblement, anæsthesia and vesical troubles, which disappeared under the influence of suggestion. The disturbances due to neuritis gradually grew better. The above-named symptoms were considered to be hysterical because the loss of power was out of proportion to the slight degree of neuritis, the electrical reactions of the muscles being normal, and on account of the influence of suggestion. The other cases

included hysterical retention of urine in connection with cystitis, cured by suggestion; attacks of hysterical sleep in the course of a case of neurasthenia; and hysterical symptoms complicating endometritis.

Clinical Notes of Cases of Hysteria reported by Dr. Ghilarducci from the wards of M. Charcot.[47]—In the first, there were attacks of typical sensory epilepsy; numbness invading one half of the body, including the tongue; paralysis of the limbs, motor aphasia, and mental disturbances, chiefly loss of memory. The family and personal history of the patient pointed to tuberculosis, and at one time the diagnosis of a lesion of the cortex was made. The signs of hysteria were the occurrence of hystero-epileptic attacks and the presence of the permanent stigmata.

In the second patient, attacks resembling those of Jacksonian epilepsy from a lesion in the neighbourhood of Broca's convolution occurred, accompanied or preceded by paresis and tremor of the right arm and motor aphasia, ending in general contracture of the muscles, loss of consciousness, and biting of the tongue.

The third patient, besides well-marked symptoms of hysteria, suffered from attacks beginning with cramps in the calf of the left leg, a feeling of cold which passed rapidly over one half of the body, and was followed by tonic and chronic contractions of the two limbs most marked in the lower extremity.

In the fourth case, the symptoms took the form of localised epileptiform convulsions, resembling the evolution of cerebral syphilis. There are also accounts of a case of "hysterical apoplexy" *i.e.*, of hysterical hemiplegia with unusual characters. All these cases are reported in great detail, and the points in the differential diagnosis of each of them fully entered into.

M. Babinski,[48] in another paper on organic and hysterical contractions, observes that according to Brissaud and P. Richer hysterical contractures present the closest analogy to spastic contractions due to sclerosis of the lateral columns; in both there is muscular rigidity, together with exaggeration of the tendon reflexes, and sometimes ankle clonus. The author, however, believes that apart from any other symptoms in other parts of the body, indicating either hysterical or organic disease, the characters of an hysterical contraction are, in themselves, pathognomonic. As a point of distinction in organic hemiplegia the author lays stress on a feature of the paralysis in the upper extremity that Dr. Buzzard has previously pointed out. Dr. Buzzard[49] says: "In true hemiplegic contracture . . . you cannot, by any amount of force, straighten the whole limb at

one moment. If you straighten out the fingers the wrist remains rigidly flexed. Bring the metatarsus into a line with the forearm by extending the wrist, the fingers will *ipso facto* become rigidly flexed." In hysterical contracture the forearm, hand, and fingers can be brought into the same plane. Of equal importance is the exaggeration of the tendon reflexes in organic disease. M. Babinski thinks that a manifest and unilateral exaggeration of tendon reflexes in a case of spasmodic hemiplegia indicates, as a rule, that the motor troubles are not hysterical; this holds good in a case of paraplegia, if the bilateral exaggeration of reflexes is very pronounced and accompanied by ankle clonus.

The reasons for the above statements are: (1) In a very large number of cases of spontaneous or provoked hysterical contractures in hypnotised subjects, the author has found no notable modification of the tendon reflexes. (2) The independence of hysterical contraction and of exaggeration of the tendon reflexes was well shown in a case of right hemiplegia. In this patient an hypnotic, *i.e.*, hysterical contracture could easily be provoked on the left side, and this contracture did not modify, in any way, the tendon reflexes. On the right side an hypnotic contracture could be provoked by suggestion in the lower extremity, in which the motor power was preserved relatively to the arm, but this contracture was much less intense than that evoked in the opposite limb. It was, however, impossible to modify the condition of the right upper extremity, in which there was marked paralysis, either to augment the rigidity already existing in it by hypnosis, or to produce an hysterical contracture. (3) In a case of tabes with absent knee-jerks, pronounced contracture of the lower limbs could be produced during hypnosis. Since observations, contradictory to these statements, have been made by competent observers, is it possible that two forms of hysterical contraction exist, one without and one with exaggeration of tendon reflexes? The author thinks that the last condition must be altogether exceptional, although he will not directly deny its existence; perhaps the difficulties and pitfalls which lie in the way of exact observations on the state of the reflexes may account for some of the discrepancies in the statements. He points out that exaggeration of reflexes may be apparent only, and be counterfeited by other reflex or voluntary movements, excited by the percussion of the tendons. Difficulties of diagnosis may obviously be great where hysterical is associated with contracture of organic origin, but in these cases the latter cannot be very marked.

With regard to the nature of these contractures M. Babinski

remarks that relaxation is as active a change as contraction of a muscle; although certain features of these contractures correspond to those of voluntary contraction, yet the spasmodic rigidity of organic disease cannot be considered as identical with prolonged muscular contraction, for it is impossible to voluntarily produce, even transitorily, a state of muscular rigidity with the objective characters of organic rigidity (contracture). As to hysterical contracture, however, it is possible to produce, during a short time, a muscular rigidity which corresponds exactly in objective characters to hysterical contracture, and in ordinary hysterical cases a contracture may succeed a voluntary contraction and the inequality of the duration of each constitute the only difference between them. A patient, with hysterical contracture, departs from the normal in the length of time he is able to maintain a contraction, or rather in that he is unable to relax his muscles after contraction. Comparing hysterical contracture and paralysis, in the former the patient is unable to induce relaxation, in the latter contraction of the muscles affected.

Hysteria and Disseminated Sclerosis (Löwenfeld).—With regard to the diagnosis between disseminated sclerosis and the forms of hysteria which resemble it so closely, the author remarks that the hysterical tremor and movement generally continues during rest, and is not a true tremor occurring only with voluntary movement, but one that merely *increases* during movement, shows great alterations in intensity, and may suddenly disappear. In hysterical speech affections the monotonous scanning speech of disseminated sclerosis is not observed, but rather a form of stuttering. If hemiplegia is present it is characterised as hysterical by the accompanying hemianæsthesia, glosso-labial spasm, absence of facial paralysis and of the signs of descending sclerosis. The nystagmus and pallor or atrophy of optic disc, common in disseminated sclerosis, are absent in hysteria. See also Buzzard.[50]

Prof. Grasset[51] reports the case of a woman, aged 23, suffering from hysteria and disseminated sclerosis, in which the symptoms of disseminated sclerosis were nystagmus, scanning speech, characteristic tremor of the limbs and head during voluntary movement only. Exaggeration of the knee-jerks and ankle clonus. No affection of sensation, except slight diminution in the right leg. The special senses were normal except vision. As to the latter there was (1) considerable, but irregularly, concentric contraction of both visual fields—the outline differing from hysteria in its marked irregularity; (2) no dyschromatopsia; (3) atrophy of optic discs consecutive to neuritis.

Five weeks after admission the patient had two hysterical convulsive attacks, caused by a strong emotion. Besides the defective sensation in the right lower extremity, other hysterical symptoms now present were hysterogenic zones over the ovaries and under one breast, and loss of the pharyngeal reflex.

About eight months later the patient was, without warning, seized with serious bulbar symptoms and died in a short time. At the autopsy the bulb and cord only could be examined; the bulb presented a "compact sclerosis," with some more distinct points of better defined sclerosis; in the cord were several characteristic patches of sclerosis, most numerous in the cervical and lumbar enlargements.

Professor Charcot[52] relates the case of a young man, aged 31, who discovered whilst in his bath that his left lower extremity was markedly wasted, the foot and the leg being chiefly affected. His astonishment and fright was so great that he had an hysterical attack shortly after noticing this. No loss of power or affection of sensibility had occurred to lead him to think there was anything wrong with his leg. On examination, the circumference at lower third of thigh, right 38 cm., left 34 cm.; at middle of calf, right 38 cm., left 31 cm. There was some spasm of the extensors of the toes. The atrophied muscles were those of the foot, leg, thigh and buttock; they were the seat of fibrillary tremors, but there was only very slight loss of power in them, and the electrical reactions were in all normal. The patient could walk perfectly naturally. The knee-jerks were exaggerated; no ankle clonus, no alteration in the colour, temperature or sensibility of the limb. The patient came of a family prone to hysterical affections, and had shown signs of hysteria himself. It should have been said that the muscles of the right shoulder and upper arm also showed commencing atrophy, fibrillary tremors, and exaggerated tendon reflexes. Professor Charcot points out that the above symptoms with absence of change in electrical reactions in any of the affected muscles exclude lesions of the nerves, cord or brain; but rather consort with the hypothesis of purely functional or dynamic lesion. Hysteria is capable of causing such muscular atrophies with characters that simulate atrophies of spinal origin, and which follow the course usual in other hysterical manifestations appearing and disappearing with them. Facts of this kind have been recorded by Kahlkof working under Seligmuller, Babinski, Blocq, Debove and others. These hysterical muscular atrophies coincide frequently with paralysis or anæsthesia of the affected

limb; they are sometimes developed in a few days, and after a variable time pass off either slowly or quickly. They are not accompanied by fibrillary contractions; recent observations, however, have shown that hysterical muscular atrophy may be attended with fibrillary tremor, and the seat of the atrophy with the exaggeration of the reflexes sometimes met with in hysteria make the resemblance to atrophy of spinal origin a strong one. These cases are, however, exceptional, and an electrical examination will settle the diagnosis, for so far the reaction of degeneration has never been found in hysterical atrophy. Such being the features of hysterical muscular atrophy, there is nothing to absolutely gainsay the dynamic or functional origin of the muscular atrophy in the above case.

VII.—Papers on the Traumatic Neurosis.

Dr. Strümpell,[58] in discussing the traumatic neuroses, says that for some years, especially since the establishment of legislation for compensation for accident, physicians have had frequent opportunities of observing patients who complain of a group of undoubtedly nervous symptoms as the result of an accident. Before the accident they were quite well, and in one group of these cases the connection between the injury and the nervous disturbances is undoubtedly clear. Whatever be the nature of the injury, the character of the illness can be estimated by the ordinary means of general and local diagnosis.

Another group of cases presents very different features. The injury has not fallen on a part where important nerve structures lie, or has not been a very severe one, so that a severe effect on any part soever of the nervous system is not probable. The kind of pains and nervous disturbances suffered by the patients are not of the kind that occur in connection with grave nerve-lesions. The symptoms are many of them of a general nature, spread beyond the injured part, and certain symptoms are always absent which are met with in organic nervous affections. The characters of the cases in this group are distinct from those produced by grave injuries of the nervous system, and correspond to those found in functional nervous affections. As it is the custom to call these functional affections "neuroses," this group of cases has been called "traumatic neurosis," in order on the one hand to emphasise the connection with injury, and on the other the functional nature of the disturbance.

The term *traumatic neurosis* was never intended to be used to indicate a special and fixed disease, but rather as a short designation provisionally acceptable for a group of morbid conditions, whose clinical symptoms and pathogenesis must first be established by special investigations. The use of the expression the *traumatic neuroses* should have indicated that the conception of a special traumatic neurosis was in no wise permissible. The group of symptoms are in fact not by any means always the same. With the view of a provisional sifting and grouping of the cases, the author has separated them into (1) cases with clearly local nervous symptoms (pains, paralyses, contractures, anæsthesias, &c.), (2) cases with undoubted general symptoms of disturbance of central nervous system, without in any way intending to make "local traumatic neurosis" a special disease. He then briefly describes the form of disease which is commonly meant when a "traumatic neurosis" is spoken of.

The patient has suffered from the effects of a fall or blow for a longer or shorter time, and although the immediate effects of the injury have long passed away, he has never regained his former health. He attributes his altered physical condition directly to the injury. Though a blow on the head or chest may cause pains in the head or chest respectively, yet the pains are never entirely localised, and correspond to no special nerve distribution. With the pains is a corresponding loss of motor power, which renders all active bodily work impossible. Together with loss of physical strength, there is incapacity for mental exertion, insomnia, loss of appetite, palpitation, sense of pressure in the head, &c. "The more the patient is questioned as to special symptoms, the more complaints are obtained in answer." The objective signs of disease are, in comparison with the subjective, insignificant. Only after close examination of all the nervous functions does one hit upon those symptoms which Oppenheim first laid stress upon as "the objective signs of the traumatic neurosis," and the word "objective" must be taken *cum grano salis*. The chief objective symptoms are the various anæsthesias and hyperæsthesias of the skin, affections of the special senses, contraction of the visual fields, &c. They are real and important symptoms, but depend on the "state of consciousness" of the patient, and their significance must on that account be altogether different from the independent and objective signs of organic nerve disease. The skilful physician will be able to recognise the real origin of these symptoms from the character and extent of the affections of

sensation and of the special senses. The contradictory character, want of uniformity, and variability of the symptoms is only striking to the unlearned. And yet it is quite intelligible that the statements of different physicians over the same morbid state should be at variance. For the nature of the clinical investigation has a great influence on symptoms of psychic origin. For instance, he who constantly searches for contraction of the visual fields will be more likely to meet with it frequently than the man who doubts the existence of this symptom. Many so-called *objective laws* of hysterical phenomena rest on the operation by suggestion of the method of investigation.

Whether the term *traumatic neurosis* is retained or abandoned, and replaced by the names *traumatic hysteria* and *traumatic neurasthenia*, is indifferent so long as one holds fast to a sharply defined definition of what is understood by this name. Practically it appears reasonable to provisionally retain the name traumatic neurosis as a collective term, whilst allowing that it does not exist as a specific disease.

Dr. Strümpell raises some specially important points in the differential diagnosis. Fr. Schultze has pointed out that many patients refer their troubles to an injury, whilst they are suffering from a general morbid process such as arterio-sclerosis or alcoholism. Further, the decision that a material nerve injury of some kind or other has not taken place in some of the cases of predominant local symptoms is by no means an easy one, especially in the presence of fractures, deep wounds, &c.

Perhaps certain reported cases of *traumatic neurosis* in which such anatomical changes as *optic atrophy*, *fixity of pupil* were observed, must be referred to chronic organic changes produced by the injury (commotionstörung). The writer has observed neuroses which went on to a well-marked progressive paralysis. He has seldom observed intentional simulation, and points out that simulation in traumatic neurosis is not at all easy. How should a simple workman, totally ignorant of the observations of the Salpêtrière school, be able to simulate loss of smell and taste, and hemianæsthesia? The constancy of the symptoms would imply a special pathological process. In diagnosis the author would put more weight on the *tout-ensemble* of the disease, and general mental impression than on single symptoms, and thinks that isolated persistent objective symptoms, even when found, have only a relative value. But it must never be forgotten that all symptoms of disease dependent upon psychical conditions follow quite different rules from those which have an anatomical basis.

If the reality of the disease is to be put in doubt on account of the instability of certain symptoms, and of the exaggerations of the patient, then half one's private patients must be simulators for the same reason.

However clear the diagnosis of single cases may be clinically, their forensic consideration is generally difficult. How far the insurance companies must be liable, how far allowance must be made for the subjective psychical factor which plays such a great *role* in the causation of the neurosis is questionable. Treatment should be taken in hand at once, as the bad prognosis and want of response to treatment in this disorder results partly from the fact that the patients go for months or years from one doctor to another, from one court to another, till the symptoms of their malady become fixed and graven in their innermost being. The rule for treatment is "Principiis obsta"!

Dr. Ch. Vibert,[34] as medical expert to the tribunal of the Seine, has had the opportunity of making numerous observations on the results of severe accidents. He has been able to follow up a certain number of cases for several years, and has learnt from them that the disorders which develop consecutively on an accident may, after an interval of apparent good health, go on developing in intensity even to a fatal termination.

Five persons in the Charenton railway accident, who had no external wound nor fracture, had punctiform hæmorrhages over the whole or half of the upper part of the body, together with deep congestion of the face, and sub-conjunctival hæmorrhages. Similar appearances were found in persons with fractured skull, or extensive wounds of the head, and the author is inclined to attribute them to lesion of the nervous centres, probably the bulb, but has no direct *post-mortem* evidence of this. The lower extremities are much more frequently damaged than the upper in railway accidents. Insomnia, muscular tremor, a highly-emotional state, or one of hebetude, stupor, or a semi-conscious condition are the nervous conditions which most frequently directly follow the accident, and as a rule disappear in a fortnight's time. This nervous shock plays no part in the development of the grave cerebral disorders which follow an accident unless the brain itself has been injured by concussion or commotion, except that sometimes excessive emotion causes more profound and lasting troubles than those mentioned above.

The most common general symptoms are loss of appetite, and various digestive disturbances, asthenia, depression, loss of flesh, anæmia, and profuse sweating on any exertion.

In the case of a diabetic who sustained contusions of the head, and a severe wound of the leg in a railway accident, the glycosuria was not aggravated.

Cases of direct or indirect injury to the lungs and stomach are given in detail.

Cerebral troubles occasionally appear after an interval of some weeks of health has succeeded the disappearance of the first symptoms of shock, but in the majority of cases they come on immediately after the accident. These disorders are *intellectual*, as loss of memory, loss of the power of attention, melancholy, apathy, indifference, irascibility, emotional excess, headache, often vertigo; all aggravated by intellectual work, emotion, and especially by alcohol.

Disorders of special senses.—Ears: Tinnitus aurium, sometimes deafness, inability to bear any noise. Eyes: Photophobia, paresis of accommodation. Taste and smell are rarely affected.

General sensation.—Subjective sensations in the limbs; the author has not observed anæsthesia.

Motion.—Intermittent muscular twitchings, subsultus tendinum provoked or aggravated by emotion. General loss of muscular power, giving rise to feebleness of gait. Affection of the sphincters is not present. Various disturbances of the general health accompany or result from the cerebral disorders.

The author believes that general shaking (commotio cerebri) is answerable for the early symptoms, and that the later ones arise from a meningo-encephalitis which results from material lesions, *e.g.*, contusions, ecchymoses, sanguineous effusions occupying solely or exclusively the surface of the cerebrum, and other parts of the encephalon. He therefore considers the prognosis a grave one in such cases, and cure little likely to result; in any particular case it is often impossible to give an exact prognosis. As in many cases in which cure follows after some weeks, or two or three months, the same symptoms are present as in those which do not recover, it is generally necessary to wait six to eight weeks, and watch the course of events before giving a prognosis. In cases where the cerebral troubles are profound, and accompanied by severe general symptoms, the prognosis is necessarily bad, and it is equally so in the cases where the symptoms come on late after a period of apparent health.

As regards simulation, the author lays stress upon the fact that in his experience the intellectual troubles and purely subjective symptoms are never grave without at the same time a corresponding gravity of the general bodily symptoms (objective)

mentioned above. Exaggeration may be suspected, if there is no proportional correspondence between the two.

Rarely cerebral affections may develop secondarily to those of the cord.

With regard to injuries of the cord in railway accidents, the author has little new to add to the well-known works of Erichsen and Page.

The author pleads for the retention of the term "traumatic neurosis" on practical grounds, independently of any theory which may be attached to it. The expression conveys clearly the dependence of the symptoms on an injury. He distinguishes three forms: (1) the hysterical (2) the common form, called by French authors hystero-neurasthenic; (3) a form in which one special symptom predominates.

(1) Cases in which an injury has brought about the development either of hysteria pure and simple, or of nervous disorders for the most part of clearly hysterical nature. The author states, that although he has examined a large number of injured persons he has never met with a case resembling the type described by Charcot and Guinon; and only one case of hemianæsthesia.

(2) Variety in which paralyses, contractures, anæsthesias, convulsive attacks either do not exist at all or only in mild degree; the chief symptoms being those of a cerebral state which has often been described, *i.e.*, depression of spirits, restlessness, irascibility, incapacity for mental or physical exertion, loss of memory, asthenopia and insomnia, with attacks of nausea, faintness and palpitation, together with various disturbances of the heart and of the digestive organs.

(3) In which one particular symptom predominates, *e.g.*, cardiac symptoms or dyspnœa, or rarely delirium.

Etiology.—The traumatic neurosis is produced by an injury acting in conjunction with an emotion—terror. Traumatic hysteria may be excited by an insignificant wound, but although this is proved, cases of hysteria pure and simple are only exceptionally met with in the traumatic neurosis; cases of the latter have a more definite etiology in arising after an injury of special nature, which appears to play as important a part as emotion. In the author's experience of twelve years as a medical expert in such cases, he has never seen, except in one woman, the group of symptoms which make up the traumatic neurosis present in persons who have met with all sorts of accidents, wounds from knives and revolver bullets, partial strangulation and attempts at suicide in various ways. Out of a much smaller number (about

400) of cases of victims of railway and carriage accidents, explosions and falls, he has met with 100 cases of the traumatic neurosis. In many accidents of the former category the emotional disturbance must be almost as great as in the latter. The nature of the injury is, however, different, because in the second class of accidents there is a more or less violent shaking of the nervous centres. This commotion is certainly present in persons who have received a violent blow on the head, and these are precisely the cases which are affected in greatest proportion by the traumatic neurosis. The group of symptoms of the traumatic neurosis corresponds to the description that has long been given by surgeons of the nervous symptoms which follow head injuries. It may often be very difficult to decide whether the trouble is purely functional or is due to a gross lesion of the brain or its membranes. In those patients who have not sustained a blow on the head there is reason to believe that there has been a severe commotion of the brain, spinal cord or nervous system generally.

Emotion as a factor.—Most persons affected with the traumatic neurosis have been more or less terrified at the time of the accident; sometimes the terror has been so great and the wound so small as to make the former appear most important; but the author thinks that the influence of emotion has often been exaggerated. For those persons most terrified at the time of the accident are not those who suffer most frequently from the traumatic neurosis, while those who do so suffer have often displayed little emotion at the time. Perhaps the effects of emotion are aggravated when there is also more or less physical "commotion" of the brain. Again, the symptoms of the neurosis may appear in cases where from the sudden nature of the accident there can have been no time for emotional disturbance. The author does not consider that neuropathic tendency or predisposition plays an important part in the development of the traumatic neurosis; evidence of such a tendency is frequently absent, and the coincidence of injury and evident emotion is sufficient to produce the symptoms.

The statements in this paper appear to be fairly borne out by the details of numerous cases which are given.

Prof. Pel[55] reports the case of a carpenter, aged 27, with no family or personal history of disease; but with a large head from rickets or hydrocephalus. After a heavy fall (? on the head) which caused loss of consciousness, he complained of persistent localised pains in the back of the head on the left side, and subsequently of giddiness on movement, together with an over-

powering tendency to sleep. The left half of the back part of the skull was tender to percussion. There was paresis of the right half of the body and left facial nerve. Power of vision was deficient. All these disorders were permanent and did not change, but in part were slowly progressive. Sensation was everywhere normal. The tendon reflexes in both lower extremities were exaggerated. Standing and walking was impossible, but when lying on his back in bed the patient had unimpaired movement of his legs. As the patient had a small polypus on the right vocal cord, which was caught between the cords on phonation, it was removed without any difficulty under cocaine. Immediately after the operation speech was lost, and attempts to speak only resulted in a wider separation of the vocal cords from each other; the levators of the epiglottis were also paretic.

Three weeks later there was noticed for the first time right hemianæsthesia, hemianalgesia, hemianosmia, hemiamblyopia with loss of taste on both sides. Two months after the operation the patient could only whisper feebly, and could scarcely put the mouth and lips into the positions necessary for forming letters. Subsequently he had two hystero-epileptic attacks.

Complete paralysis of the adductors of the vocal cords after such a harmless procedure as the removal of a small laryngeal polypus could only be explained as a "local traumatic neurosis," and strengthened the diagnosis already made of the condition previous to operation as that of traumatic neurosis. The appearance of right-sided anæsthesia and bilateral loss of taste without increase of the previously existing right-sided paresis, together with hysterical mutism and hystero-epileptic attacks, clearly indicated that the case was an example of the classical male hysteria or traumatic hysteria of French writers.

As to the further history of the case, none of the various modes of treatment tried, including hypnotism, produced any effect; the man died of a double catarrhal pneumonia which was fatal within a few days of the onset, and was a further proof of the small resistance offered by such a patient to the attacks of acute disease. Except for hydrocephalus the brain was healthy, presenting no abnormality which could account for the symptoms observed during life.

In conclusion, the author emphasises the fact that functional nervous disorders may follow not only on an injury, but the performance of an operation or any method of local treatment of an irritable organ, and this should be borne in mind, since the possibility of such an occurrence is apt to be forgotten in the

present day, when attention is directed chiefly or only to the material consequences of surgical procedures.

Dr. Morton Prince[95] observes that great difference of opinion exists as to the prognosis in the traumatic neurosis, some holding that the tendency of all cases is towards recovery, and that at an early period; others that the pathological condition disappears if at all only after a prolonged duration. He therefore records two cases of twenty-nine and one of twenty-eight years' duration, which belong to the so-called hysterical type, are of the male sex, and have, so far as can be judged, maintained their original form without the development of secondary pathological consequences.

The first patient was struck by a piece of shell which did not injure the skin beyond a black and blue mark below the left shoulder blade. This left side was "paralysed immediately after the accident, and has remained so ever since." Dr. Prince saw him in 1886 and in 1891. At the first visit he suffered from anæsthesia of the left arm and leg only, at the second this extended over the whole left side of the body, except the face and palm. There was also left hemiparesis, and some deafness, loss of taste and of smell on the left side. Knee-jerks were both within normal limits. The patient stated that his condition varied from day to day. In the second case, one of gunshot wound, the ball entered the left arm between the upper end of the belly of the biceps and the deltoid tendon passing through the bone. There was, on examination in 1886, complete paralysis of all the muscles of the arm and hand from the shoulder down, including the deltoid. There was also slight loss of sensation over the limb; no atrophy or degenerative rigidity or contracture of the muscles, which were softer than those of the left arm from disuse. In the third case there was decided, but not absolute paralysis of the muscles of the left hand and arm, including the deltoid, without atrophy, rigidity, or exaggeration of the deep reflexes; there was also profound, but not absolute anæsthesia over the limb. The condition came on after recovery from twenty-four hours' loss of consciousness, consequent on injury from the wind of a round shot which passed between the extended arm and the body; it had remained the same until Dr. Prince examined him in 1886, and again in 1892 it was found unaltered.

The author concludes that the picture presented resembles that of functional disease, and that this diagnosis would have been made in cases presenting similar symptoms immediately after an

injury; he sees no reason to question the diagnosis because of the long persistence of the symptoms.

Traumatic Neurasthenia.—Dr. Löwenfeld's experience in South Germany is that the working classes are affected by neurasthenia and not by hysteria, as the consequence of accidents. This traumatic neurasthenia is distinguished by the cause only, and not by the symptoms; but symptoms of cerebrasthenia predominate. There is no hereditary taint in these cases; often, but not invariably, an interval of freedom from symptoms intervenes between the accident and the onset of neurasthenia. Bouveret attaches more importance to nervous shock than to physical injury, but in many cases the latter is equally potent. The sufferers from traumatic neurasthenia never regain their full powers of work, but after a time are capable of doing lighter work.

The Traumatic Neurosis.—Löwenfeld remarks that Oppenheim and Strümpell are the chief advocates of the view that there is a distinct disorder of the nervous system arising from injuries—the "traumatic neurosis." Charcot held the extreme opposite view, that such cases were instances of hysteria — of "traumatic hysteria." Contrary to the statements of French authors, the existence of a specific "traumatic neurosis" was never generally believed in in Germany. The author was the first to indicate the existence of "traumatic neurasthenia," and of German authors, the first to show that a specific "traumatic neurosis" did not occur. Injuries may give rise, according to individual predisposition, to various nervous disorders. Chorea, epilepsy, paralysis agitans may follow an injury as well as hysteria and neurasthenia, yet in the great majority of cases, one of the two latter diseases is found or a combination of both— hystero-neurasthenia. These latter cases correspond to Oppenheim's "traumatic neurosis."

Other factors in the causation of such cases, besides the mental effect of the injury, must be considered. Charcot and Oppenheim consider the mental effect as the most important, and they are justified in so doing in a large number of cases, as is shown by the fact that the extent and intensity of the resulting nervous disturbance bears little or no relation to the severity of the injury. Yet wherever there has been a mechanical shaking (commotion) of the central nervous system, the influence of this on the head and spinal cord, must be of, at least, more importance than that of terror. Schmauss showed experimentally that commotions of the spinal cord which were without any gross change in the organ, resulted in destruction (degeneration) of nerve fibres. The neurosis under discussion does not appear at

once, but is gradually developed from molecular disturbances. In slighter degrees the effect of injury shows itself as a functional change, an exhaustion of the nerve fibres, and this exhaustion may, in severe cases, lead to their degeneration. Sudden powerful shocks (commotion) in the central nervous system in man may produce similar effects, and it is therefore not correct to neglect the mechanical causes in traumatic nervous cases so much as Charcot and Oppenheim have done.

In severe injuries, without, however, actual damage to the nervous centres, there are the depressing influences of pain, with attendant loss of sleep, appetite, and fresh air, and anxiety with regard to future means of subsistence.

The fact that slight injuries, quite unimportant in themselves, may result in neuroses, can be explained only by taking into consideration the individual predisposition. The combined effect of bodily injury and mental shock may even in a perfectly sound man give rise to nervous disorders. In the cases in which moderate injuries to the body without mechanical effects on the central nervous system cause lasting neurotic disturbances, there must have been an antecedent neuropathic tendency. The more pronounced this neuropathic disposition, the more trivial may be the injury capable of giving rise to subsequent nervous disorders. This predisposition may be inherited or it may be acquired from alcoholism, masturbation, former accidents, injurious influences of mode of life or occupation. Lastly, the nature and seat of the injury may have an influence on the seat and character of the nervous troubles.

Dr. Knapp,[57] in discussing the question of "simulation in traumatic nervous diseases," thinks that the tendency has been to exaggerate the frequency of simulation in nervous affections of traumatic origin. The reproach of simulation is sometimes made because the examining physician is a general practitioner without any special training in or knowledge of nervous disease; to such a man a patient suffering from astasia-asbasia, a patient, for instance, who might be able to hop but unable to walk, would seem to present a clear case of simulation, yet an experienced neurologist would admit the genuineness of the condition. Again in hysteria, the nature of the conditions is often curious and improbable, *e.g.*, monocular diplopia; there may be extreme amenability to suggestion, so that the manipulations of a prolonged examination may accentuate or diminish the symptoms present; the influence of stimuli is often very marked. "When we prove that a patient, under a powerful stimulus, can do what

she says she cannot, that her pain is most marked when her attention is called to it, that her condition varies from day to day . . . . . we do not disprove the existence of hysteria or of neurasthenia."

Certain symptoms are valuable; contraction of the visual field can with difficulty or not at all be successfully simulated. Anæsthesia, if the boundary is well defined, may be safely assumed genuine. If anæsthesia is simulated an *unexpected* painful stimulus will soon detect it. Differences in the skin reflexes in hemianæsthesia will establish the existence of anæsthesia beyond dispute. A *persistent* rapid pulse is of positive value, as is probably also an acceleration of the pulse produced by pressure on painful regions. The author thinks that the simulation of one of the more complex forms of traumatic nervous disorder cannot be successfully maintained through a long medical examination by a skilled neurologist. In fifty consecutive cases of all kinds which he has examined with reference to determining the degree of injury and the amount of compensation to be claimed, in only two was simulation probable, and one of these patients was a child.[58]

VIII.—General Features of Neurasthenia (Löwenfeld).

Stigmata of Neurasthenia.—Certain symptoms occur again and again with monotonous regularity in cases of neurasthenia. They are almost constantly present, and form the ground work of the disease or "stigmata," to use Charcot's expression. They are limited in number. Dizziness and pains in the head, inability for mental work, disturbed sleep and irritability of temper characterise the cerebral, pains in the back and weakness in the limbs, the spinal form of neurasthenia. Add to these palpitation, nervous dyspepsia and sexual weakness, and the list of typical symptoms around which others are grouped in varying number is complete.

Many symptoms were formerly allotted to hysteria, hypochondriasis and melancholia, that later observation has shown to be really due to neurasthenia, and in hypochondriasis a number of neurasthenic symptoms occur which are merely concomitant with the hypochondriacal symptoms and are really due to the accompanying neurasthenia. The author has found in his study of neurasthenia that the symptoms are not only subjective, but

that there are a number of objective signs. The complaints and personal history of the patients afford an insight of especial value into their personality. He distinguishes clinically four types of sufferers.

Clinical Types.—(1) Those patients who preface their tale by saying that they will make a short statement of their complaints in order not to take up the physician's valuable time. This is the prelude to an account in fullest detail of their medical history from their earliest years. This is Charcot's "*l'homme aux petits papiers.*"

(2) Those who are more reserved, wish the physician to find out for himself what is the matter with them, and have an innate suspicion that their bodily state and nervous symptoms are not in accord; they do not wish to prejudice the physician by unsolicited statements.

(3) Those who say that they enjoy absolutely good health except for one symptom, *e.g.* insomnia.

(4) Those who from anxiety or dejection often experience a certain constraint in speaking out to the doctor. Neurasthenia from excessive onanism, from a fall in social position, and traumatic neurasthenia.

Intellectual Disturbances.—Intellectual disorders are of three kinds:—

(1) In the least severe cerebral exhaustion leads to degradation of intellectual capacity through slight failure of memory, lack of power of attention, and ready fatigue after mental exertion.

(2) In more severe cases besides quantitative loss in the intellectual sphere, not only is long-continued mental work impossible, but the general usefulness of the sufferer is impaired.

(3) In the most severe cases the capacity for mental work sinks to *nil*, the sufferer leads a purely vegetative life. In practice these three grades pass imperceptibly one into the other.

The emotional disturbances are well known. Those in the sphere of will are briefly indecision, dislike of change, but also instability in opinion, neglect to maintain personal honour, or to fulfil a distinct promise, fear of physical discomfort, and the condition described by Möbius as "akinesia algera." The character undergoes deterioration, but Arndt's view of moral insanity in such cases is based on a misconception.

Uncontrollable thoughts (*Zwangsdenken*) divided into uncontrollable (*a*) ideas (in the narrower sense); (*b*) actions; (*c*) sensations; and (*d*) emotions.

(*a*) Questionings of *cui bono*, over the nature of things and the existence of God; fear of mental unsoundness, to be distinguished from the hypochondriacal form; syphilophobia, misophobia, "délire du toucher" (admirably described by George Borrow in the character of the literary recluse in "Lavengro"), fear of robbers, of thieves, of fire, &c.; fear that they may be impelled to commit suicide; echolalia, coprolalia. In many of these cases the mental symptoms predominate over the others, throwing them into the background.

(*b*) Uncontrollable sensations affect chiefly the head and sexual organs. They play an important part in the causation of many cases of agoraphobia.

(*c*) These, in the form of uncontrollable fears or anxiety, constitute a very important class. Such conditions of unfounded irresistible fear or dread are met with in a quarter of the cases. They completely overmaster the patient in the attacks, but are generally confined to one particular set of conditions, and they affect persons to whom in all other circumstances fear is unknown.

The best known is the often described agoraphobia; in the worst cases of this affection the patients are unable to leave their homes, but in many the companionship even of a little child, or the fact that another person is walking in front of them, enables them to proceed with confidence. There are other dreads too numerous to describe; we note as of special importance the idea of inability to perform some special action, and especially some public function, which may prevent priests, professors, advocates, or statesmen from carrying on their avocations. There is ever a morbid fear of becoming possessed by one of these dreads.

The author describes an "angor nocturnus" in which patients wake up in a state of terror, which lasts from five minutes to half an hour, is attended with shivering and sweating, and followed by great prostration. Feelings of undefined horror, of impending misfortune, "a horror of great darkness" frequently afflict neurasthenics. Various corporeal disturbances such as dimness of vision, pulsation of cephalic arteries, inability to swallow, dryness of the mouth, changes in the respiratory and cardiac rhythms, motor weakness, trembling of the legs or whole body, heavy sweats, retching, desire to urinate or defæcate, may accompany and often follow the psychical troubles, and entail a state of exhaustion which lasts for some time, and render comprehensible the determined avoidance by the patients of the conditions likely to induce an attack.

Such conditions arising in mentally sound persons from circumstances inadequate to produce them in the healthy may be traced: (1) often to an attack of real illness or of faintness, or qualmishness affecting the patient in church, in court, or in the street, and the dread of a recurrence leading to persistent agoraphobia or similar state; or (2) the first onset may have been a sudden dread of being overtaken by illness during some public function; (3) they may come on suddenly and inexplicably. The surroundings of the attack then become fixed in the memory, and when the patient is again in the same place act as a stimulus to provoke a fresh attack. An auto-suggestion of incapacity, a fear of breaking down thus occurs, and it is important to remember that it may be overcome by another (fresh) suggestion —for instance, if the patient can be made confident that the thing he considers out of his power can be accomplished and that he will not break down. Similar conditions of fearfulness, or want of confidence are probably experienced by nearly everyone at some time in their lives, but they persist and master the neurasthenic because he cannot recognise their groundlessness and throw them off as the healthy man does. In the consideration of these uncontrollable thoughts, actions, and conditions of dread are to be noted: (1) the marked disturbance of the bulbar, and especially of the vasomotor centres. The repeated alterations in the calibre of the vessels in vasomotor disturbances, thus frequently recurring, may lead by interference with cerebral nutrition to psychical disturbance. There is a marked state of abnormal reflex psychic irritability. (2) The resemblance of these neurasthenic brain conditions to the hypnotic state. (3) Loss of control or lack of will power in neurasthenic patients is important, as it prevents them from resisting the development of these "fixed" or uncontrollable ideas.

Some authors have regarded these peculiar mental states as abortive monomanias, or paranoia. The author agrees with Koch who thinks that they cannot be considered as mental disease because they leave the patient untouched in his general mental processes, perceptions and relations with the external world; they dominate but do not falsify his mental processes, and rather hinder than favour the development of a psychosis. The author has found that they are especially frequent and obstinate in cases of hereditary neurasthenia, but in no way confined to these.

Vertigo.—Attacks of vertigo with subjective sensations of sinking, staggering, falling, &c., are very common in neurasthenia;

the more severe attacks are often accompanied by nausea, obscuration of vision, noises in the ears, and pallor. Paroxysmal states of mental confusion or stupefaction with numbness, coldness and weakness in one or both extremities on one side of the body also occur. These attacks come on without definite cause, but some are connected with stomach disorders.

Subjective Painful Sensations.—Various sensations are felt in the head, of pain, emptiness, opening and shutting, pressure, &c. (cephalic sensations of Gowers). Pain most commonly occurs in the back or sides of the head, sometimes over the eyes, and often as a characteristic feeling of a tight band encircling the head (*casque neurasthénique* of Charcot). There may be hyperæsthesia of the scalp over the seat of pain. These cephalic sensations occur in 75 per cent. of cases, vary in nature and intensity in different cases and in the same case from time to time. The causes are inherited tendency to neurasthenia, attacks of meningitis in childhood, injuries to the head attended with shock or fright, onanism, alcoholism, and long-continued mental over-exertion. They are much more common than the often described rachialgia or spinal irritation. Spinal pains often alternate with pains in the head; when originating in sexual excess or abuse they are attended with mental states of peculiar anxiety. Sensory disturbance of vision apart from migraine is rare, but feelings of numbness, stiffness, or loss of power in the tongue are not uncommon, generally affect circumscribed parts of it, and may be accompanied by difficulty in speaking. Pains are common in other parts of the body, and may simulate the lightning pains of tabes dorsalis, but are rarely so intense, and are more fugitive in character, rapidly changing their situation. Further cases of neurasthenia are met with in which there are general neuralgic pains. The common feeling of weakness or weariness in neurasthenia is not due to motor weakness; it is felt most before the patient stirs. Weariness and muscular weakness are two symptoms of neurasthenia which often occur together, but in the author's opinion are not causally connected.

Hyperæsthesia.—Hyperæsthesia may be general or affect circumscribed areas of skin. The underlying organs, *e.g.*, breast, testicle, or muscles may be tender to pressure. In traumatic neurasthenia, hyperæsthesia and hyperalgesia are common, and may last after the other effects of the accident are gone. Sometimes there is increased power of localisation of sensations over the hyperæsthetic areas. In traumatic neuroses Mannkopf's symptom (increased rapidity of pulse on pressure over the tender

areas) is sometimes present, but observations are still required to show whether this holds for ordinary cases of neurasthenia. On the other hand anæsthesia is rare, and then only affects small areas of skin; the anæsthesia found in cases of hystero-neurasthenia is to be attributed to the hysterical element in the case.

Attacks resembling Focal Sensory Epilepsy.—The author relates a case of a man, age 50, affected with neurasthenia for twenty years, and who for twelve years had suffered about once a year from attacks in which feelings of pricking or numbness began in the occipital region, and spread over the left side of the head and face to the left arm, left side of the body, penis and left leg; they lasted for three minutes, and were followed by a feeling of weakness in the same parts for a quarter to half an hour; examination detected some weakness of the affected parts. The absence of any sign of organic disease, and of any permanent nervous trouble during so long a period shows the functional nature of the attacks. He has met with attacks of similar sensations in other patients limited to one arm or leg, or to the arm and leg on the same side, and often attended with cephalic sensations. The resemblance is very striking to the similar attacks which occur in migraine. Migraine is not a direct symptom of neurasthenia, but the latter has a striking influence on the severity, frequency, and duration of the migrainous attacks, which are aggravated should a condition of neurasthenia supervene in a patient subject to them.

In one case the attacks above described, which resembled those of sensory Jacksonian epilepsy, ceased when the neurasthenic condition improved.

Eyes.—Neurasthenic asthenopia, or irritable eye. There is a difficulty of fixation, the object fixed soon becomes indistinct, and if fixation is persisted in, shooting, stabbing, or boring pains in the eye come on. Long standing cases of neurasthenia may suffer from pains during rest or on movement of the eyes, and occasionally from double vision. These visual troubles are not connected with errors of refraction.

The author finds that contraction of the visual field rarely occurs apart from asthenopia, and that it is, as a rule, absent in neurasthenia. Charcot disputed the existence of contraction of the field in neurasthenia. Oppenheim attached great importance to it in the diagnosis of the traumatic neurosis, but a number of other observers deny its occurrence in such cases. Wilbrand's[98] observations, however, which are the latest, correspond with Oppenheim's, and he points out that the divergent opinions may have arisen from different methods of observation. An objective

symptom in the traumatic neuroses is that known as Förster's typus. The essential point in this contraction of the visual field is this, that if an object be brought from the periphery to the centre of the field, it is seen further away from the centre than when it is moved in the contrary direction. The pupils may show excessive mobility or alternating dilatation and contraction independent of light or accommodation. Inequality is generally transitory, but has been observed to last for eight or ten months in a patient who recovered (there was no sign of organic disease).

Motor Disorders.—Difficulty in balancing on one leg, or in standing still with the eyes closed and the feet together has been noted, and also a *pseudo-ataxic* gait described. The author has never seen a well-marked ataxic gait in neurasthenia, and considers that its description is due to errors of observation. The gait may be unsteady from feelings of vertigo, and neurasthenic patients often deviate to one or the other side in walking, but it is never distinctly ataxic. He thinks loss of power is often largely of sensory origin. Other motor symptoms are a tremor like that of Graves' disease; fibrillary twitchings of the muscles, especially in the eye muscles, or the tongue; involuntary short contractions of single muscles or groups of muscles, especially of the thumb muscles, which may be sufficient to move the arm or leg, and prevent sleep or cause a restless condition.

The superficial and deep reflexes are as a rule increased. The author emphatically states that the knee-jerk is never lost in neurasthenia; with one or two exceptions, this is the general opinion.

Speech.—Disturbance of speech commonly takes the form of paraphasia, inability to recall the appropriate word or phrase, tendency to change or put in a wrong word, *e.g.*, to say good morning instead of good evening. Often the interchanged word has no connection with, or even the opposite meaning to that intended. Spasm of the pharynx or a feeling of stiffness in the tongue may render speech difficult or indistinct. Unimportant words may be rapidly repeated several times without stammer, or there may be stammering. All speech troubles are worse after excitement or over-exertion.

Neurasthenia Cordis.—Neurasthenic affections of the heart are frequent, occurring in more than 50 per cent. of cases. The pulse rate in neurasthenia is, as a rule, from 80 to 100 per minute, but may be persistently over 100, with or without attacks of tachycardia, in which it reaches from 180 to 200. These attacks may come on after excitement, overwork, the use

of coffee, or some error in diet, or without any assignable cause, and are in many cases ushered in by the presence of subjective feelings in the heart, and accompanied by faintness, feeling of impending death, excessive anxiety and necessity of emptying the bladder and bowels. The attack leaves behind it pronounced weakness of the heart, and a condition of general exhaustion, which only disappears after some hours. The author has not met with the severe forms of tachycardia in neurasthenia described by Bouveret, but does not deny the possibility of their occurrence. These attacks differ from true angina pectoris in the majority of cases in the lesser intensity of the symptoms. Slowness of the pulse (40 or 50 to minute), is only occasionally met with. Very seldom weakness of the heart, as a result of over-strain, anxiety, alcoholism, &c., is so pronounced as to give rise to fears for the patient's life. The patients sometimes suffer from "hyperæsthesia of the heart and vessels," in which they are conscious of the heart labouring, and the blood circulating down to the finger tips. Flushings, blushings, especially of the face, disturbances of vasomotor innervation, lead to temporary or lasting dilatation of vessels. Frequently changes in the temporal arteries, by which they become more prominent and tortuous, are seen in young men of from 20 to 30 years of age. This pre-senile change is to be regarded as a sign of neurasthenia.

Nervous Dyspepsia.—Two forms of nervous dyspepsia occur: (1) patients in whom the general nutrition is unaffected; (2) those who waste, lose colour, and show signs of general malnutrition. This difference is by no means solely connected with the duration of the illness. In the mild form there are the usual symptoms of flatulent dyspepsia, the patients do not completely lose appetite, but are afraid to eat for fear of exciting pain. Other troubles, such as interference with heart-beat and respiration may perhaps be referred to pressure from a dilated stomach, but are also in part due to reflex influences on the bulbar centres. In the severe form, the wasting and loss of strength in connection with digestive troubles may go so far as to cause fears for the patient's life. Complete loss of appetite and disgust for food is even more than pain a cause of insufficient nourishment. Further observations are needed to reconcile the conflicting statement as to deficiency or excess of HCl. secretion in the neurasthenic stomach. Mathieu attaches more importance to impaired motility of stomach than to anomalies of secretion, but the subjective pains and discomfort bear no relation to the insufficiency of motor power.

With regard to intestinal troubles (enteropathies), the chief are excessive production of gas and constipation or diarrhœa. The author enumerates the disturbances of general health, and of the bowels which follow from constipation, and is also inclined to attribute stomach dyspepsia largely to constipation, because of its preventing the proper emptying of the stomach, and of the relief which is often given by a free purge at the onset. Later in the course of neurasthenia the constipation increases, and spontaneous evacuation of the bowels is lost altogether; they become distended with gas, and later still attacks of diarrhœa from irritation alternate with constipation, and the large intestine passes into a chronic catarrhal condition. The author notices amongst the visceral neuralgias attacks of pains in the liver which resemble biliary colic. There is generally a striking increase of the knee-jerks; the attacks come on at remarkably regular intervals. In diagnosis, the fact that no inflammatory complications are found after the attacks have occurred for years, is important, and treatment directed to the nervous system is more efficacious than a course at Carlsbad.

Urine.—With regard to the urine the author notes the frequent occurrence of phosphaturia and of oxaluria, as dependent on disturbance of the general metabolism; he distinguishes two kinds of oxaluria: the one, in which the crystals are minute and do not give rise to local irritation, the other, in which they are larger and occasion pains or discomfort in perinæum and urethra, with frequent calls to micturition. He thinks no special diagnostic or ætiological importance attaches to the presence in the urine of the so-called urethral casts (*Urethralfäden or Tripperfäden*) which are made up of a ground substance of mucus with layers of epithelial or round cells in varying proportion, and often contain spermatozoa or oxalate crystals. These casts come from the urethra, or the prostate, and result from chronic urethritis.

Sometimes polyuria is met with, lasting for months or years and accompanied by excessive thirst, loss of appetite and symptoms of nervous dyspepsia. The author thinks thirst is the primary disturbance and polyuria the secondary.[1] In three cases of neurasthenia the author has found a trace of albumen in the urine, the amount varying slightly with the patient's general condition. Although it cannot be denied that a slight albuminuria may be dependent on neurasthenia, this conclusion should only be arrived at after every other cause for the albumen has

[1] See paper above on "Hysterical Polyuria."

been excluded. In badly nourished anæmic persons the albuminuria may be a consequence of the general condition.

The idiosyncrasies so frequently met with in neurasthenia with regard to tea, coffee, tobacco, wine, food, and certain drugs are attributed to auto-suggestion.

Clinical Varieties of Neurasthenia.—According to the symptoms present neurasthenia has been divided into the following groups:—(1) cerebral neurasthenia or cerebrasthenia; (2) spinal, myelasthenia, spinal irritation; (3) cerebro-spinal or general; (4) cardiac neurasthenia (nervous heart weakness); (5) gastric (nervous dyspepsia); (6) sexual; (7) neuralgic form; (8) hemineurasthenia; (9) hystero-neurasthenia. These numerous divisions are to be explained by the fact that few patients complain of all symptoms, one especial group predominating; this is especially the case in nervous dyspepsia, in sexual and in cardiac neurasthenic conditions. In the latter condition, symptoms of general neurasthenia may be present, but those relating to the heart cause the patient so much anxiety that he forgets to complain of his other sufferings.

(1) By cerebral or spinal neurasthenia it is not intended to imply that the nervous exhaustion falls solely on the brain or spinal cord; the connection between the two is so close that, as both clinical and experimental observations show, neither can be affected alone. But in individuals the resistance to morbid influences varies in different centres, so that cases occur in which signs of cerebral or spinal disturbance predominate. In many cases of cerebrasthenia physical condition and muscular strength are unimpaired. In the so-called psychical form the symptoms vary; in a great number of neurasthenics psychical changes (in volition, sensibility, and idea) are present, and these psychical anomalies stand in very varying relation to the signs of general cerebral exhaustion, which latter may be in no proportional intensity to the increased emotional irritability, feeble volition, and hypochondriacal or melancholic conditions present. Marked changes in temper, disposition, and emotional excitability may occur with unimpaired power for mental work and for sleep. Symptoms of loss of control (*Zwangsaffekte*) may be absent in cerebrasthenia or be marked whilst the mental symptoms are slight.

(2) *Myelasthenia.*—Two symptoms predominate: (*a*) pains in the back; (*b*) weakness of the limbs, especially of the legs; they are not necessarily in proportionate relation to each other. Numerous paræsthesiæ are felt in the legs, spermatic cord,

bladder, and prostate. The author distinguishes the following clinical varieties.

(*a*) Spinal irritation; feelings of stiffness and pressure in the back with pains radiating to the seat, private parts, and limbs. These pains have a very depressing effect. (*b*) Neuralgic form; in which pains of lancinating character affect chiefly the extremities. (*c*) Sexual. (*d*) Pseudo-ataxic form. (*e*) Form affecting the cervical portion of the cord.

(3) In sexual neurasthenia the author lays special stress on the evil influence of masturbation, on account of its frequency, and also because it is often begun at an early age when the central nervous system is not fully developed. Those cases of neurasthenia are alone to be considered of sexual origin in which disturbances of the sexual organs are the only, or the predominating, symptoms. Cases of neurasthenia due to these injurious influences do not always take the form of sexual neurasthenia.

Cases of sexual origin are divided into:

(*a*) Those in whom the functional disturbances are wholly confined to sexual centres in lumbar cord. Damage to the sexual functions may reach the highest grade without affecting other parts of the central nervous system. In some patients spermatorrhœa and impotence do not interfere with bodily or mental faculties, whilst in others of neuropathic tendency general disorders are entailed.

(*b*) The symptoms of sexual disorder are associated with symptoms of neurasthenic affections of other parts of the nervous system, generally brain and cord.

(*c*) In which sexual disorders come on secondarily to general neurasthenia, *e.g.*, as result of a fever, and increase the nervous exhaustion from causing frequent seminal emissions. An irritable condition of the lumbar cord may, however, have been induced by inheritance or sexual abuses, and be first brought out by the action of a cause entailing general nervous exhaustion.

(4) *Hereditary Neurasthenia.*—According to Beard, a chief characteristic of hereditary neurasthenia is its obstinacy. Levillain divides these cases into three chief varieties:—(*a*) Neurasthenia complicated by hereditary symptoms (phobias, tics, *délire du toucher*); (*b*) Neurasthenia, brought about by accidental causes, which develops into hystero-neurasthenia; (*c*) Neurasthenia of distinct hereditary origin.

(*a*) The author says that in one group of cases it cannot be shown that the neurasthenic condition arises through an inherited neuropathic tendency. Other causes sufficient to produce it in

those of healthy inheritance are present. In a second group the causes are complicated, and the influence of heredity is not always clear. In a third and very large group the influence of inheritance is manifest, neurasthenia developing under circumstances insufficient to produce it in the healthy. In a fourth group neurasthenia comes on without any definable reason. These individuals show signs of an abnormal condition of the central nervous system in childhood, such as headache, convulsions, attacks of migraine, night terrors, and somnambulism. They are often quick intellectually, but break down in an unaccountable manner on any extra strain. Any intercurrent malady leaves behind it an increase of the nervous condition. In the strict sense of the term hereditary neurasthenia should be confined to this last class.

There are no pathognomonic signs of hereditary neurasthenia, but uncontrollable ideas and emotions (Zwangvorstellungen and Zwangsaffekte), and the so-called phobias (agoraphobia, &c.) are especially frequent, attain a greater degree than in other cases, and persist after the other symptoms have been removed. Psychical anomalies are also most marked in this form. There is a "circular" or recurrent form in which the same symptoms observe a periodic recurrence, each period lasting for some days.

Cause and Prognosis of Neurasthenia.—Neurasthenia has no fixed course on account of the individual variations in the cause of the illness, in the circumstances and mode of life of the patient, and in the treatment adopted. One characteristic is the frequent occurrence of variations in the course of the disease, of remissions, of return without assignable reason of symptoms which have disappeared, much to the discouragement of the patient. When this occurs without assignable cause, it should be borne in mind that disturbances of menstruation in women and of the sexual functions in men are apt to be concealed from the physician. This variation in symptoms is especially frequent in hereditary neurasthenia.

Neurasthenia does not, with certain exceptions, shorten life, and it interferes with rather than altogether puts a stop to the duties of life. As a rule, the disease responds well to therapeutic measures, cure, or, at any rate, more or less relief being obtained. The patient's surroundings and mode of life are of most importance in the prognosis. If a man has to continue working day and night for his bread, or is exposed to constant anxiety in his business; if a wife or mother must go on nursing a husband or child in spite of exhausted strength, then the dis-

order will naturally be increased; if, on the other hand, the patient can be put under better conditions, recovery can be secured.

The duration of the illness is of more importance in prognosis than the degree of nervous exhaustion. Symptoms of short duration are easily cured, and *vice versâ*. As a rule, hereditary neurasthenia is especially obstinate, and a complete cure is altogether exceptional, but great improvement is possible. Of most influence in retarding recovery are insomnia, nervous dyspepsia, and nocturnal emissions.

There is no proof that organic disease of the nervous system can proceed from neurasthenic conditions, but the commencement of general paralysis often resembles neurasthenia, and here there are two possibilities, either that neurasthenia may lead to changes which pave the way for the further action of alcohol and syphilis on the brain, or that the neurasthenic condition is connected with the commencement of the changes of general paralysis. Not very rarely neurasthenia passes into a psychosis of functional character, most frequently into melancholia.

Myelasthenia does not give rise to tabes dorsalis; neurasthenic cardiac disturbances rarely, if ever, lead to actual heart disease, and the attacks of tachycardia are never fatal.

Neurasthenia may lead to alcoholism, morphinism, cocainism, &c.

On Diagnosis in Neurasthenia.—(1) The patient's statements must never be solely relied upon in diagnosis. A complete picture of the case must be formed out of the patient's antecedent history, the cause and development of his troubles, together with the results of a thorough physical examination.

(2) On the mental side of chief importance are failure of capacity in different fields of intellectual work and defective memory, especially for recent events. Special groups of incidents may be forgotten, and fictitious events unwittingly substituted for them. Fixed hallucinations do not occur; uncontrollable ideas (Zwangvorstellungen) do not interfere with general mental operations, nor falsify ordinary perceptions, but persist in the mind as a sort of foreign body.

(3) Pains in the head are seldom of great intensity or long duration. Percussion tenderness over the head is absent, except in traumatic cases.

(4) No persistent paralytic conditions in the limbs; this is especially true of the facial and oculomotor muscles; such paralysis is against the presence of neurasthenia.

(5) Persistent alterations in speech are absent.

(6) Inequality of pupils is found but with good reaction to light and accommodation.

(7) The knee-jerk is never lost; diminution of knee-jerks is also unusual.

(8) That an illness is of neurasthenic character will be the more certain the longer it has lasted without the development of symptoms of organic disease.

(9) From another point of view the changing character of neurasthenic symptoms is important, but this point must not be pressed too far for in certain organic diseases the symptoms may vary or disappear, and in neurasthenia some, such as the cephalic sensations, loss of mental capacity, sexual disorder, and nervous dyspepsia are very persistent. A sharp distinction of neurasthenia from hysteria is not practicable, because the symptoms of the former are so frequently associated in the same patient with those of the latter. There is, however, no difficulty in distinguishing the symptoms which belong to each neurosis if the so-called hysterical stigmata, *e.g.*, anæsthesias, hemianæsthesia, paralyses, contractures, different forms of convulsions, attacks of sleep, and hysterogenic zones are borne in mind and their presence sought for. No reliance is to be placed on the often stated inconstancy or fleeting character of hysterical symptoms.

IX.—Treatment of Hysteria and Neurasthenia (Lowenfeld).

The general aim of all treatment is of course to remove the cause, but in removing the local disorder which may have preceded for some time the general disturbance of the central nervous system one cannot always relieve the latter, and in many the former is an expression of the general nervous condition. If the changes in the nerve centres which form the basis of neurasthenia and hysteria have, whatever be the original cause, been kept up for some time, the removal of the cause does not insure the return of the centres to the normal state. That depends on the duration of the changes in question; on the constitutional powers of resistance of the nervous system, and on the presence or not of an inherited or acquired neuropathic constitution, and takes place if at all only very gradually. In many cases a perfect restoration of the central nervous system to its normal condition is no longer to be attained.

As regards diet the author is not in favour of the vegetarianism that has been recommended for these disorders, but thinks a mixed diet in easily assimilable form at not too long intervals is best. He restricts the use of coffee in cases of heart weakness. When neurasthenia has come on as the result of deficient nutrition, as the body-weight increases and nutrition improves the nervous symptoms dwindle, but in most cases no such result is to be anticipated, wasting being a secondary result of the nervous disorder. If persistent alteration in the composition of the gastric juice be present, in cases of increased secretion of acid the income of fats and carbo-hydrates must be restricted whilst the digestion of meat goes on well; bread containing dextrin for starch and the use of such fats as butter are, however, desirable. In deficient acidity digestion of proteid can be carried on in the duodenum, if not in the stomach, and meat need not be cut off, but merely restricted in quantity. The use of alcohol must be decided by the individual habits and the indications in each case. Patients who suffer from tendency to swooning and disagreeable sensations in the head with flushings of the face are better without it.

Occupation.—As a rule, the occupation must be interrupted in neurasthenia for a longer or shorter time, especially in the case of occupations which entail much anxiety or over-fatigue, or which fall heavily on one nervous function. The worst cases must give up their work permanently, but it is not generally wise to completely forbid the occupation altogether; it is better to restrict the amount of work so as to bring it within the patient's powers.

In hypochondriacal cases it is particularly important that they should have some occupation to keep their attention off their complaints. In pronounced myelasthenia stop physical work; in brain-exhaustion, on the other hand, it is within limits useful; but *excessive* muscular exertion is a hindrance to recovery in brain-exhaustion also.

Sexual intercourse must be regulated, as sexual excess is highly injurious. No general answer can be made as to the desirability of marriage.

Medicines.—Medicines can only palliate the symptoms and not cure them. The bromides, preferably the mixed bromides, take the first place, and are especially useful in conditions of nervous irritability, but are injurious in cases where weakness and exhaustion predominate. Protracted administration of bromides is inadvisable. The suitable dose is 20-30 grains, or 52 grains per diem. Bromo-camphor is useful for nervous palpitation: ·1 to ·2 grammes three times daily.

Quinine is useful, strychnine not; atrophine is good in spermatorrhœa or too frequent nocturnal emissions. Iron is often abused in treating states of nervous weakness, but where there is anæmia, or disturbance of nutrition is the primary and nervous disorder the secondary condition, it is useful. Valerian is of service in psychical excitement, tendency to anxiety, &c. Narcotics in order of usefulness are codeina, sulphonal and paraldehyde. A drug that deserves notice but must be used with care, and is not to be given in heart-weakness, is hyoscine; especially useful in hysterical convulsions, with almost instant effects, in small doses of $\frac{1}{10}$-$\frac{3}{10}$ milligramme. Of antipyrin, antifebrin and phenacetin, the first is the best in headache, especially in migraine, in neuralgic affections generally, and in "cephalic sensations." Phenacetin resembles the bromides in action.

Change of Air.—The beneficial influence of change of air and surroundings in these neuroses is well-known; in old protracted cases so far as change of air can do good it must act by bringing the patient under climatic conditions entirely different from his habitual ones, and this is especially to be attained in sea and mountain climates.

Sea.—Many cases do badly at the seaside suffering from increased irritability, loss of sleep and appetite, and general weakness. Sea air is especially indicated in cases of exhaustion from excessive brain work and similar causes in persons of good digestive powers, and in conditions of nervous weakness with insufficient blood formation. It is not suitable for severe cases of hysteria, and patients with marked psychic disturbance.

High Altitudes.—In a large group of cases mountain residence is useful, but not as the sole means of cure. Severe cases of old standing will not be improved by mountain, more than by any other air. However favourable the influence of the air may have been during the residence of the patients at a high altitude the old troubles will come back sooner or later after their return to their homes. Even permanent mountain residence may without other aids, bring about no change in the condition of the nervous system. For this group, mountain air is to be regarded as an after-cure, to consolidate and perfect the benefit received from other modes of treatment. The therapeutic effect bears no relation to the actual height. Improvement shows itself in decreased irritability, increased mental capacity and muscular power, in improved sleep, digestion, and sexual power, and in relief of palpitation.

Hydropathic Treatment.—The author attaches especial value

to hydrotherapeutic measures. One of the most useful is the wet pack. On rising from bed the patient is enveloped, all except the head, in a wet sheet; the attendant rubs the body all over for one minute, and then rubbing in a dry, warm sheet follows. The effect of this treatment varies with the temperature of the water (50° to 81° F. is the most generally useful), and the roughness of the towel.

The general effect is a stimulating and invigorating one. Another method is the ordinary wet pack which may be followed by frictions in a cold bath for three to five minutes. He also especially recommends a bath, taken as follows. The patient sits up to his middle in water, whilst an attendant dashes the water over his back and chest; he then rubs himself, or is rubbed all over with a loofah or rough towel, or in cases of paraplegic weakness the legs especially are rubbed. After this, the whole body is immersed in the bath; and, finally, a cold douche thrown over the neck and back. Duration of whole, six minutes. Temperature of bath 72° to 86° F.; of douche 8° to 10° F. lower.

Sitz baths are often useful, especially in habitual constipation, but contra-indicated in irritable conditions of sexual organs.

Sea-bathing.—Sea-bathing is contra-indicated in thin anæmic persons who cannot stand the loss of heat entailed by it, also in cases with either insomnia, much neuralgia, paræsthesiæ, palpitation, convulsive attacks, tendency to head congestion or in severe myelasthenic conditions. *Warm* sea-water baths have not received the attention they deserve, and often give good results in increased appetite and tissue change, and improved nutrition. They are partly sedative, partly stimulating.

Electrical Treatment.—Löwenfeld advises the application of a weak constant current to the head by very large electrodes for one or two minutes in cases of brain exhaustion, and in "cephalic sensations." As a guide to the management of the applications no unpleasant sensations should follow the sitting, if the manner and kind of application has been suitable to the case. He also attaches much importance to galvanization of the spine, not only in myelasthenia, but also in general neurasthenia. Currents to pass transversely from sternum to dorsal spine, or from navel to lumbar spine. The electrode on the spine is to be moved upwards and downwards, keeping it longer on any painful spot or over the part of the cord most affected. Electrodes to be 5 cm. by 10 cm., current strength 2-5 ma., duration five minutes.

In all cases of nervous disturbance of heart's action, abnormal

cardiac sensations, præcordal pain and anxiety, angina pectoris and in nervous asthma, galvanization of the vagus, by placing the +ve pole on inner border of sterno-mastoid above the manubrium and the —ve below the lower jaw, with a current of ½-2 ma. for 1½-3 minutes is useful.

Galvanization of the lumbar part of the cord is advised for sexual disorders; and in some cases, in which there is no chronic urethritis an intra-urethral electrode is to be applied to the prostatic part of the urethra. The author endorses the use of general faradization as employed by Beard and Rockwell, and finds it especially useful in hysteria and neurasthenia with symptoms of profound exhaustion and in neurasthenia with defective nutrition, poor appetite and emotional depression.

Psychical or Moral Treatment.—(1) Negative, consisting in prohibition of mental over-exertion, removal of injurious influences, such as emotional excitations exerting an unfavourable effect, the provision of sufficient employment for mind and body adapted to the patient's strength, and in certain cases isolation.

(2) Positive. Here it is of prime importance that a thorough explanation of the nature of his complaint, namely, that it depends on functional disturbance and not on organic disease, should be given to the patient. He should be encouraged to look for recovery. These disorders are aggravated and maintained by the patient's fears for the future, fears of impending brain and cord disease. Dispel these fears and a material step towards recovery is gained. If he knows the nature of his disease the sufferer will not experience fresh alarm at every fresh symptom. At the same time he must be impressed with the necessity of self-mastery, of putting forth the whole power of will to overcome his pains and tendency to excessive anxiety, and must be made to understand that he is considered to be really ill. To laugh at these patients, to rally them on their healthy appearance, and tell them that their sufferings rest on imagination is only to court mistrust. Educated persons, now-a-days, readily grasp the distinction between "organic" and "functional" nervous disease; the uneducated may be helped by illustrations. Then the source of the nervous disorder must be indicated in order that the patient may avoid it in future. Patients should be placed amongst congenial surroundings. They gain ease and relief of mind if the whole mode of life, meals, drink, exercise, and occupation is regulated by the physician, who must not only give directions, but see that they are obeyed. Every detail of the treatment advised, however disagreeable, must be scrupulously carried out, and hydrotherapeutic measures are

a useful means of discipline to this end, and give the patient more endurance and greater confidence. The author endorses the value of the Weir-Mitchell treatment in the more severe forms of hysteria and neurasthenia, but thinks that the number of cases in which the full treatment is necessary is a limited one.

Prof. Erb,[59] in remarking on the undoubtedly great increase of the neuroses during the last half century, observes that if this increase is to go on at the same rate the outlook for the future welfare of the nations is a gloomy one. But there are, fortunately, still a great number of persons unaffected by these changes who must replace the worn-out brain-workers, and there is reason to think that increased knowledge of the danger will result in pure, healthy conditions of life, in new methods of education and care in training, by means of which the workers will be prevented from falling into the same state. The peasant, rather than the artisan class, are likely to supply the future brain-workers free from nervous conditions. Then, by careful and judicious training, the nervous system may be educated, developed, and adapted to meet without suffering injuriously the increased demands made upon it by the complex conditions of modern life.

With regard to the prevention of the neuroses judicious training of the nervous system and of the body as a whole is most important. It is chimerical to expect that the intermarriage of neurotic persons can ever be prevented. Infants and children must be better cared for, more judiciously trained, both mentally and bodily. At school, athletic exercises in the open air must be insisted upon, and sufficient time allowed for them. Undue pressure must be avoided, only necessary things taught, and non-essential requirements be given up. Care of the body and a thorough physical training is most essential, and a right proportion between work and leisure, of which latter a due share should be spent in the open air in physical exercises, is as necessary for men engaged in sedentary occupations entailing much brain-work as it is for children and youths at school. Girls must be educated primarily for being healthy wives and mothers. Restriction in the use of such poisons as tobacco and alcohol must be practised.

In a paper on the subject of treatment, Dr. P. Blocq[60] observes that when hysteria was considered to be of uterine origin, treatment was directed to this organ; afterwards it was treated as a general neurosis by anti-spasmodics; now the tendency is to mental and moral treatment, as the opinion gains ground that it

is a psychical malady. Of the correctness of the view that hysteria is a peculiar modification of psychical action the author entertains no doubt. He tabulates the modes of treatment as :

Psychical by (*a*) isolation. The *rationale* of this treatment is the separation of the patient for a variable time from his customary surroundings. To be effectual it must be as complete as possible. The best conditions are obtained in a hospital or hydropathic establishment. Further experience has fully confirmed Charcot's teaching that there is no more effectual treatment for hysteria than isolation. Isolation acts partly passively in putting an end to the conditions which engender hysteria, actively in being exactly opposed to the psychical disturbance which characterises the neurosis. The fact of removing the patient puts a stop to the series of causes—for instance, the excessive solicitude and anxiety of the friends, their grief or terror at some of the manifestations of the neurosis, or the presence of other relations affected with nerve disease—which have aided the development and contributed to the persistence of the malady. Isolation hinders that want of mental concentration, that distraction which in M. Janet's view plays so important a part in the development of some of the symptoms; whilst on the other hand it substitutes the idea of cure for the hysterical manifestation with which the patient's mind is occupied.

(*b*) *Hypnotism.* — Theoretically the tendency to increased "suggestibility" in hysteria, *i.e.*, for sensory impulses of any kind to evoke, by distubance of the normal processes of association, abnormal and persistent motor effects, would seem to contra-indicate the employment of hypnotism because it also renders the patient more amenable to influence by suggestion, but practically it has a limited application for the relief of serious symptoms, in which other therapeutic measures fail. The inconvenience of increasing the patient's amenability to influence by suggestion is more than counterbalanced by relief of aphonia, paralysis and contractures by means of hypnosis. A certain number of hysterics cannot be hypnotised; in others the symptom is relieved for a time, but soon returns, or is sometimes replaced by a more disagreeable one. In any case the rules laid down by Professor Pitres should be followed. (1) In all cases to obtain the patients' consent, and to wait until they themselves suggest a trial of hypnosis. (2) Never to hypnotise a woman except in the presence of others. (3) Only to give suggestions useful for cure.

Transference.—This method, in which by the application of a

magnet unilateral anæsthesia, contracture or paralysis is transferred to the opposite side, is sometimes useful, for the symptoms when transferred are less tenacious, more amenable to treatment, and may even disappear completely after transference.

Metallo-therapy.—The application of metals acting by indirect suggestion is occasionally undoubtedly instrumental in curing various hysterical manifestations.

Hydro-therapeutics, of all external applications, are the most useful, and to be most often had resource to. Preferably cold douches 13-18° C., by a jet thrown first on the body, ending on the feet for 15-20 seconds once or twice a day followed or not by rubbing. The wet sheet may be employed where this method cannot be followed out.

Electricity.—Static electricity is most generally suitable.

Massage and gymnastic movements are both of great service. The above methods can of course be applied whilst isolation is being carried out.

Of drugs, the alkaline bromides are rarely of any use. The author, however, recommends in conditions of extreme agitation or mental excitement the bromide of camphor as follows:—Camphor mono-bromide 3 grammes, ext. of quassia 2 grammes, syrup of belladonna q. s.; divide into thirty pills; two to six per day. Valerian, valerianate of zinc, quinine may be useful, and if narcotics are necessary sulphonal and chloral.

Dr. Blocq divides hysteria into the following forms: (1) latent; (2) mild; (3) severe (grande hystérie, hystéro-épilepsie); (4) mono-symptomatic.

(1) Children, who, together with nervous inheritance, suffer from troubles such as somnambulism, night-terrors, attacks of violent temper, require to be most carefully trained, as they are likely to develop hysteria in later life. Physical exercise is to be insisted upon, intellectual work and emotional excitements interdicted. Teaching should not be begun until the age of eight or nine, and then conducted in the most regular and systematic fashion. If the parents are affected with nervous maladies, the children should be sent away to school.

(2) In the author's opinion isolation is rarely necessary, and hypnotic treatment absolutely contra-indicated in this form; whilst in the third variety they both find their most important applications.

(4) Cases of mono-symptomatic hysteria are especially troublesome on account of their tenacity. All the various forms of treatment are often tried in turn without success.

Treatment of Individual Symptoms.—In the convulsive attacks it is generally sufficient to watch the patients to prevent them from injuring themselves, but if they occur in series they should be cut short either by searching for and pressing upon the hysterogenic zones, or by the inhalation of ether or bromide of ethyl.

For brachial monoplegias, Charcot's method of causing the patient to make efforts with the dynamometer several times a day, and to note the smallest progress made is to be employed. In this way almost continuous progress may be made.

For contractures, massage, the application of metals, or of a magnet are often effectual. Surgical interference is only justifiable in the case of connective tissue adhesions. In the case of hysterical anorexia, if the prescribed meal is refused, food is at once to be administered by the nasal tube.

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Reviews and Abstracts.

The Trophic Properties of Nerves.—By Justus Gaule, Professor of Physiology in the University of Zürich. An address delivered before the Physiological Section of the 63rd Congress of German Naturalists and Physicians. Translated by Mary W. Tanner.

The trophic action of nerves plays a great part in the historical development of pathology. Numerous diseases have from time to time been attributed to disturbances of it; but these theories, often asserted and often denied, have never been generally accepted because they have never been experimentally proved. Neither with irritation nor division of nerves have physiological effects been obtained comparable with the pathological conditions which had been attributed to this trophic action. In presence of these negative results pathologists were forced to choose between two alternatives. Either they denied trophic functions to the nerves in their ordinary anatomical and physiological aspect, and associated it with particular kinds of nerve-fibres specially endowed with the trophic function; or they abandoned altogether the trophic function of nerves, and gave other explanations of the conditions which had till then been attributed to it.

In the course of the last two years I have carried out a series of experiments of which I hope to show the most striking, here and in the Physiological Institute at Erlangen; and the results are no longer so negative in character. In point of fact phenomena occur as a consequence of irritation of or injury to nerves (or, more properly speaking, to parts of the nervous system, for it is the ganglion cells rather than the nerve-fibres which are concerned) which must be looked upon as far-reaching causes of disintegration of the tissues, and which approximate, more than to anything else, to the so-called trophic disturbances of the pathologists.

Doubtless, in presence of these experiments, pathology will have to re-adjust the boundaries of its experience; and I do not, therefore, think that the old views as to trophic nerves will be re-affirmed without further investigation. In the first place, it seems to me absurd to speak of special trophic nerves in the same sense as we speak of motor, sensory and secretory nerves.

I have found trophic action in both sensory and sympathetic nerves, and probably the motor participate in it. The trophic properties reside, then, in the whole nervous system, associated with the functions of its several parts, and, perhaps, connected with these, in various ways as yet unknown to us. This would suggest that the part which the sympathetic nerves play in trophic action is different from that of the sensory nerves, and this again is unlike that of the motor nerves. At all events the trophic action is not bound to any special kind of trophic nerve. It is only too evident that this latter hypothesis arose from the necessity of saving the trophic nerves at all risks. For when, in the case of nerves which could be anatomically demonstrated and physiologically examined, the result of the experiment was negative, then nerves were postulated, to which of course this experiment could have no application, seeing that they only had a hypothetical existence, and therefore could never be submitted to experiment. So pathologists will not, I hope, endeavour to explain their results in this way. But I should not wish to set aside other explanations of trophic disturbance which have, meanwhile, been attempted. Too much was hitherto attributed to the trophic properties of the nerves when it was believed that the pathological changes were caused only, and to a certain extent actually, by them. It would be absurd to suppose that external injurious influences may not act on an organism deprived of its trophic protectors; and in so far as we have learned to take these influences into account, we have added something to our knowledge which we need never forget. But we must not overlook the fact that without this weakening of the organism's power of resistance, resulting from the failure of the trophic properties, these injurious influences could not have taken effect.

When a house falls we know that it is gravity which pulls it to the ground and destroys the whole building; but we also ask what was amiss with the construction or the materials, for a well-built, let us say a "healthy," house resists the force of gravity. In presence, therefore, of the experiments which, after all, teach us to accept a trophic function in nerves, we must not rehabilitate the old views without consideration; but we shall have to ana-

lyse anew the pathological phenomena, whilst not forgetting what we have learned meanwhile.

In order to arrive at a right comprehension of the trophic action of the nervous system, I think it well to make the following remarks. The preservation of an organism depends essentially on the fact of the forces developed in it and by it being always in equilibrium with the external forces acting on it through its surroundings. As soon as this is no longer the case the external forces will gain the upper hand, and destroy or at any rate modify the organism. But the evolution of the internal energy from the latent force (Spannkraft) available in the tissues and in the food is under the control of the nervous system, which so adjusts this evolution that it holds the external forces in equilibrium. These external forces fall into two categories, not so much with regard to difference of internal character, as to the impression they make on our sense organs. One class includes rapidly varying forces, the other those which are constant and equable in their action. The first are the best known to us, physiologically at least, because our sense organs are so constructed as to be especially receptive of rapid changes. These variations are met by corresponding variations in our own evolution of energy, *i.e.*, usually by movements; just as we physically resolve the variations in external forces into movement. This play of inner and outer forces is familiar to us because it goes on under the constant control of our sense organs. We have always credited the sensory and motor nerves with the task of protecting the organism in this struggle, *i.e.*, of inducing, at every variation in the dynamic condition of our surroundings, a compensatory setting free of energy in ourselves, which brings us into a new state of equilibrium. In the cases usually cited as examples this would be accomplished by purposive defensive movements. With that idea in our minds we generally forget that there is a second category of forces by which we are equally influenced, but of which we are unconscious because they are always acting. To these belong gravity, atmospheric pressure, temperature (which is only perceived when it varies, or when it exceeds certain limits), the surrounding vapour-tension, surface-tension, certain electric conditions of the atmosphere, &c. But these forces undoubtedly affect the organism, and as certainly must the development of energy in the latter be adjusted to them in order to resist them. This adjustment must be continuous, and we may be as unconscious of it as we are of the corresponding action of external forces; but if it is to result in the well-ordered economy of the organism, it must be under the

control of the nervous system. This adjustment, then, I look upon as the trophic function of the nervous system. It is a constant, unperceived activity of the latter, about which we should know nothing if certain experiments did not show us what potent changes may result to the organism from any interference with it. If I wished to illustrate the relation which these trophic functions bear to the sensory and motor functions of the nervous system of which we are conscious, I might say that they represent the abscissa of the activity of our body, about which the movements set up by a stimulus and the other manifestations of energy fluctuate as more or less rapidly ascending and descending curves.

A connecting link between these two kinds of activities is formed, to a certain extent, by the protracted, slowly increasing influence of the nervous system on the muscles (for example, in the regulation of the capacity of the blood-vessels and of some intestines), and on the glands, &c., and we may regard these effects, according to their special character, sometimes as a new adjustment of equilibrium (as happens with a change of climate or of sea-level), sometimes as a slow variation.

Between these different functions of the nervous system there must exist internal relations, they must all be adjusted to each other; but further experiment alone can discover in what anatomical disposition, and in what physiological form these relations become evident.

At this point the following, at first sight weighty, objection might be made to these reflections. When we sever the nerve leading to an organ, say a muscle, we clearly separate that organ from the trophic adjustment, but it still remains under the influence of external forces. Now, if the previous considerations were correct, it ought to break down under this influence, and changes ought to occur in it similar to those brought about by experiments on the trophic nerve-centres. But that is not the case; the muscle does not alter, indeed in some cases it remains capable of contracting. This objection would hold good if it did not rest on a false premise, viz., that the condition of the muscle and its liberation of energy remain the same after the nerve is cut. But this is not so. I need only cite a few examples. A muscle so isolated no longer takes part in the growth of the organism, nor, if we exclude the growth, in the reparatory processes which preserve the organism from wearing out. If we injure the muscle it does not heal, and if we leave it to itself it gradually degenerates. A muscle severed from its nerve may retain its contractile power for a short time, but it

soon loses it. It is evident that the supply of material which it possesses at the moment of division may yet be used for contracting, but it is not in a condition to make good what it has used up. Just that is wanting in the muscle whose nerve has been cut which we must look upon as the peculiar characteristic of a living organism, the power of self-repair ("das sich selbst Bilden"), and its capacity for liberating energy must therefore be quite different. A whole series of chemical changes is absent when we disconnect the muscle from its trophic centre, as we learn from the study of its products of decomposition; which show, besides a few qualitative differences, such great quantitative dissimilarity that, even when the muscle is richly supplied with oxygenated blood, the products form only a fraction of those of the living muscle.

We shall, therefore, not be wrong in concluding that with the division of the nerve the muscle is put into a different condition, in which, because of its qualitatively different and quantitatively lesser metabolism, it is much less changeable and less susceptible to outer influences. No wonder, then, that a muscle still under the influence of its trophic centre shows, when the regulating function of the latter is disturbed by the experiment changes which on examination must be partly attributed to a rapid disintegration through external forces; whilst the same muscle is much less sensitive to external forces when, by dividing its nerve, it has been placed outside the influence of its trophic centre. This becomes still clearer when we consider a third condition of the muscle. A muscle with a severed nerve, is, it is true, not so quickly affected by external forces as a living one, still by degrees it is altered. It can dry up, soften, rot, &c., as muscle it will surely break down. When, on the contrary, we cut it quite out and place it in corrosive sublimate or bichromate of potash we can preserve its structure for years unchanged. What is the difference? Manifestly this. So long as the muscle is capable of metabolic action, and its substance can therefore be partly brought into a condition of decomposition, so long does it present to external forces (amongst which we must in this case reckon other life-germs), points of attack by means of which it can be destroyed; but if we completely kill the muscle, and, by hardening, bring its substance into a less changeable condition, then we render it unassailable by the outer influences to which it is usually susceptible. This should suffice to illustrate: (1) How many more points of attack the living muscle, with its constant self-repair, offers to outer forces; (2)

how great is the task of the trophic nerves of keeping it intact in opposition to these; (3) how extensive must be the changes which occur when (as at 1), the susceptible condition is maintained, whilst at the same time the regulation of the protecting force is disorganized. A complete cessation of the trophic action, which places the organ at once in the less sensitive condition, is clearly much less dangerous than an interference by which the active metabolism of the muscle is, it is true, retained, but the regulating power is disturbed.

The breaking of a valve in a moving locomotive can lead to the destruction of the engine and the whole train; but if the injury occurs while the engine is at rest, or if it stops it, then there need be no worse consequences than the breaking of a part of the machine.

Here I would draw attention to a point which is not directly brought out by the experiments in question, but which might present itself as an important factor when we have gained a deeper insight into these phenomena.

I have hitherto assumed that the trophic function consisted merely in preserving intact the whole organism and its organs in the struggle against the constantly acting forces of its surroundings; so that the breaking down of the materials present in it and the building up of new materials are accompanied by a liberation of energy which exactly balances the external forces. In point of fact the case is somewhat more complicated. An organism does not always remain the same, it gradually transforms itself. It has a period of growth, of maturity, of age, &c. During this process of transformation the relation of the separate parts is not identical. Certain organs increase in size in proportion to the others, some again decrease and disappear. We know that that occurs according to certain laws governing the whole metamorphosis of the organism. The trophic problem must therefore be extended, so that the liberation of energy in the separate organs shall be so carried on that the latter may not only maintain their status, but also remodel themselves in accordance with this general law. We must not misunderstand this in the sense that the remodelling itself is the work of the trophic functions. The causes of the transformation probably lie in the properties of the local cells; but for these to effect it without arriving at a condition of metabolism which would cause them to succumb to external influences, or would bring them into conflict with the whole vital economy of the organism, it must be under the regulating guidance of the trophic centres. This observation

is necessary to explain why the latter, under certain circumstances, have additional work put upon them, and why the results of the experiment are enhanced. I have repeatedly found grounds for this, which I hope to work out later on. I will now proceed to speak of the experiments, which to a certain extent may be considered complete.

I began with Magendie's experiment, the intra-cranial division of the trigeminal. Magendie has described fairly correctly the changes which occur in the cornea after his experiment, and the long discussion which followed it would have triumphantly proved the trophic functions of the nerves if (1) the result had not been so uncertain; and (2) if Magendie himself, like most of his followers, had not fallen into the error of looking on the ensuing changes to a certain extent as actual operations working from within outwards; and (3) if the experiment had not stood too much alone. I have taken up the experiment again, and have endeavoured to make it conclusive. That is not such an easy matter, for as the knife works out of sight in the cranium, it is hardly possible, even with much practice, to know exactly where we divide the nerve, and the result depends on the position of the cut. But we can ascertain this by dissection after each experiment, and can compare the effects which occurred before the death of the animal, and then we learn that trophic changes occurred each time the cut was made in the ganglion, and did not occur when it was made outside it. Now, with the section outside the ganglion, the nerve fibres only were divided, while with that in the ganglion, not only were these divided, but the ganglion cells were injured; so we come to the important conclusion, which was a guide to me in my later experiments, that *it is the injury to the ganglion cells which interferes with the trophic action*. To this Eckhard has raised the objection that trophic changes occur when the trigeminal is divided above the ganglion, and he described a method of lighting on the nerve where it leaves the pons. I have followed his method, but on making a microscopical examination of this portion of the trigeminal, I found (in agreement with all the known facts of brain anatomy) that there are many ganglion cells in it. So in this case also it is these which are injured. If, on the contrary, we light upon the part which is seen under the microscope to be free from ganglion cells—that above the Gasserian ganglion, but not so near to the pons—then we get no trophic disturbance. This I considered as settling the point upon which the certainty of the result depends. As to the second point, there were weighty

objections to the view, shared by Magendie himself and many others, that the changes in the cornea were to be looked upon in some measure as an inflammation actually originated by the trophic nerve. It is easy to show that external injuries are concerned in these changes, and that the inflammation may be almost entirely avoided by excluding them. Thus an opposite party arose who attributed the consequences of dividing the trigeminal to outward injuries alone. Because they found that the latter had some influence on the condition of things, they took them for the sole cause. At the same time, they quite forgot that an eye whose trigeminal—or rather whose Gasserian ganglion—has not been divided (for example, the other eye of the same animal, which has been exposed to just the same injuries), remains unaltered, while the eye on the side operated on is destroyed. Some change must have occurred in the latter as compared with the eye not operated on, by means of which external forces have got the better of it. There remained still another explanation. The eye of the side operated on is anæsthetic. Therefore, they said, because it was not aware of the outward injuries it could not protect itself by winking, &c. In this respect, the comparison between the eyes of which the trigeminal has been cut in the ganglion and those where the nerve was divided above the ganglion (therefore without injury to the ganglion cells), has been most instructive to me. Both eyes are anæsthetic, for sensation only depends on the nerve fibres which lead it to the brain. Neither eye, therefore, is capable of reflex action of the cornea, winking, &c. Both eyes can be injured by coarse mechanical accidents, such as a blow or the entrance of foreign bodies; and they are so injured. But when they are protected from this they act quite differently. The cornea of the merely anæsthetic eye remains almost as clear as before, but that of the eye which is also trophically injured soon becomes dim, deeper and deeper depressions appear in its epithelium, &c. The first is still quite a match for the steadily-acting forces, and is only altered by suddenly-varying actions; the latter is helpless against both. As the steadily-acting forces are always present, we need not wait, in the case of the trophically injured eye, for the occurrence of any special injury; the moment we harm the regulating ganglion cells the changes begin, and go on increasing with the summation of the causes. We can, therefore, give a different turn to the phenomena according as we remove from the cornea the action of one or other outer force. For example, we can prevent evaporation and cooling if we sew up the lid over the

eye, and so abolish the effects of these forces; but as we cannot replace the actual regulation of energy by our protective measures the cornea will never remain normal. The alteration will always be evident on microscopical examination, and in most instances will be perceptible to the naked eye, and in each case it is in the direction in which the protection we supplied was either insufficient, or itself set other external forces in action.

When we compare these facts (1) in the normal cornea, which does not require artificial protection because the regulating energy of the animal suffices; (2) in the anæsthetic cornea, which only requires protection against rapidly-varying forces (movements, &c.); (3) in the anæsthetic and trophically injured cornea, which requires protection against these as well as against constantly acting forces; we see how the view of the trophic functions which I previously explained must have followed from my first experiments. This view has also been sustained by my further researches, though I cannot say it has as yet guided me to any positive knowledge; and there arise other questions into which I shall enter later on.

The next thing was to ascertain how far other ganglion cells possessed trophic functions, and on what part of the body they exercised them. I attacked the spinal ganglia because the Gasserian ganglion is looked upon as their homologue; and began with the frog, as an animal more amenable to experiment. It soon became clear to me that to arrive at any result the ganglion cells must be lighted upon somehow unawares, and I concluded from this that the instantaneous division of the Gasserian ganglion, which is inseparable from Magendie's method, was of the essence of the experiment. I could not at first explain the grounds of this otherwise than by the general reflection that the ganglion cells must be in a certain condition of life and activity to exert a trophic influence. In my latest experiments I noticed a fact which might help us to explain this circumstance. I observed in the sympathetic ganglia that a slight previous stimulus, for example, a lowering of temperature or a weak electric shock, would suffice to render them quite insensible to a subsequent experimental attack. Later on I discovered that this depends on a branch of the nerve of the ganglion, which when irritated entirely inhibits its trophic action, while after its division the ganglion is much more sensitive. Perhaps the insensibility of the exposed spinal ganglion depended on the circumstance that in the act of laying it bare, these inhibiting fibres were irritated. I emphasize the "perhaps," because fresh researches alone can inform us on this point.

With the spinal ganglion of the frog I got the best effect with small injuries made by a sharp knife in the second spinal ganglion (on the large nerve supplying the arm). The trophic changes occurred principally in the skin, and were of different degrees. Sometimes there was merely discoloration, at others there was a circumscribed bleaching of all pigment, so that white spots were formed. With more extensive changes depressions occurred in the skin, which arose from shrinking (combined with drying-up) of the epithelium; at other spots, on the contrary, there were warts, in which the layers of epithelium had increased, so that twenty and more rows of nuclei could be counted one above the other. In the most severe cases there were defects in the skin, which arose from a complete disintegration of the epidermis as well as of the cutis. I could not discover then, nor can I yet understand, why the mischief should be of such varying extent. Of course it might be that I injured the ganglion differently: but yet I had the impression that it depended greatly on the condition of the frog on which I operated. Freshly caught frogs, about twenty-four hours after capture, generally showed more serious alterations than those which had been kept longer in confinement. Neither could I make out why the changes (always with injury to the same ganglion) appeared now in one part of the skin, and now in another. Only this seemed certain, that their localization always coincided with the entrance of small nerves into the skin, and that the skin-lesions only occurred where (for example, on the breast) a cutaneous muscle was attached to the skin. Many fresh factors came here into play compared with the conditions in the corneal experiments; and we have added to our knowledge two well-ascertained facts: (1) that the trophic changes do not only occur in the sphere of the nerve whose ganglion is operated on, but over the whole body, and on the opposite side, so that the disturbance in the trophic functions must, anyhow, spread through the central organ of the nervous system to the origin of other nerves; and (2) that the division of nerve-fibres plays but a small part, for the most serious disturbances obtained when I did no more than remove a small layer of ganglion cells from the surface of the ganglion.

I was sorry to be obliged to leave the examination of the frog's spinal ganglia, for the time being, in this unfinished state. I say for the time being, for I propose to take it up again when I have gained more experience. But I clearly saw that I could not penetrate further into the question until I succeeded in

getting sure and constant results to my experiments. I therefore turned my attention to the spinal ganglia of the rabbit, which offered much more constant life-conditions, from not being subject to the alternations between hibernation and spawning, land and water life, freedom and confinement. Dogs and cats, whilst in many respects desirable, were excluded from my experiments, because it is impossible to obtain the material in anything like sufficient quantity.

The experiments with rabbits were first of all arranged with a view to testing the best methods of procedure. It appeared that from the third, fourth, fifth and sixth ganglia disturbances arose in the skin, the glands and the muscles; and these, too, on the same as well as on the opposite side in the sphere of the ganglion operated on, and also in other nerve regions. Of these disturbances I selected those of the muscles, as being the most characteristic and the most easily produced. In these experiments on rabbits I also for the first time made use of stimuli: the making and breaking of a constant and not too weak current (about four to eight Daniel cells). I found an induction current ineffectual. These stimuli were about equal in their action to injuries with knife or scissors. Irritating with an acid had the same effect. The principle seemed to explain itself as follows. A stimulation of this strength certainly sets up a change in the ganglion cells; it is also an injury. On the other hand, the injurious action derives its character either from the parts destroyed or removed, or from the remaining parts. In the first case it is a negative action, in the second it is an excitation. Now, as a partial injury to the ganglion, where portions still remain, is a more powerful agent than a total destruction of it, it follows that the excitation must be the stronger. Subsequently I used either injury or stimulus as appeared desirable for a more exact determination of the circumstances. But one thing reappeared in all the experiments; injuries and stimuli were alike powerless when they acted on the exposed ganglion. The sac in which the ganglion is enclosed must not previously be opened. Perhaps this circumstance may in the future be explained by the presence of inhibitory fibres. Further, it was shown that the change which ensues in the ganglion cells exerts its action by spreading through the posterior root to the spinal cord; for if the posterior root is divided there is no action in the muscle. But we could not ascertain how the spreading into and beyond the spinal cord occurred; and for this reason, that we were not in a position to decide beforehand which muscles would

be affected. There were many muscles in which the changes occurred, now here, now there, sometimes in numbers, sometimes singly. And so these experiments did not answer my expectations. It was only when I extended my researches to a sympathetic ganglion, the inferior cervical of the rabbit, that I was led to an experiment with such constant results that it could be used for a more searching study of the trophic functions. Here changes occurred, both with injuries and electrical stimuli, which always affected the same muscles, viz., the biceps and the psoas, which always appeared in the same part of these muscles, and were always of the same form and character. It could not, therefore, be doubted that a decided local relation existed between the ganglion and these muscles. To use a familiar expression, we may say that we have found in this ganglion the trophic centre of these muscles; for as we can functionally excite certain muscles through certain parts of the brain-cortex, so we can trophically modify certain muscles through this ganglion. I was strengthened in this opinion by an experiment, not often enough repeated it is true, in which, on irritating the first dorsal ganglion analogous trophic changes occurred in the triceps and semimembranosus. In these trophic centres the muscles must be represented in an arrangement differing greatly from their representation in the functional centres, and that is easily understood; and the fact that two such homologous muscles as the biceps on the upper, and the psoas on the lower extremity, should be governed by the same ganglion must suggest the idea that it has its origin in certain laws of embryonic development.

If the irritation of the ganglion only modified the muscle of the same side we might suppose that the paths along which the ganglionic influence passes communicate directly with the muscle as sympathetic nerves. But it as often happens that the muscles of both sides are affected, and just as often only the muscle of the opposite side, while that of the same side remains intact. This necessitates the presumption that the transference goes through the spinal cord. In point of fact a more exact examination shows that it is the branches of communication of the sympathetic with the spinal nerves, through which the action in a centripetal direction is led to the spinal ganglion, and thence on to the spinal cord. This circumstance explains how the earlier stimulations of the spinal ganglia, to which I alluded above, could originate similar, although more dispersed changes in the muscles. The effects issuing from the spinal ganglia were dispersed because they

did not invade the centres themselves, but only affected the connections between these centres and the spinal ganglia, in which connections an exchange of paths may have already occurred.

Now what is it that goes on in the muscles when the sympathetic ganglia are stimulated? Till now I have been concise in the historical account of my experiments, because I was only speaking of things which I had described in greater detail in my contributions to the *Centralblatt für Physiologie*, Bd. v., Heft 15, 16 and 22; Bd. vi., Heft 11, 13 and 25; Bd. vii., Heft 7. What I now have to communicate is for the most part new. Since my last communication to the *Centralblatt* I have succeeded in seeing the changes arise under my very eyes. I was indebted for this improvement mostly to the discovery of the inhibitory branch leading to the ganglion, to which I alluded above. After becoming acquainted with this I was able by previous irritation to render the ganglion insensible, as well as by division of the nerve to make it very sensitive. As by this means I obtained better control over the stimulus, and could apply it when and how I liked, I ventured to expose the biceps on one or both sides by a slit in the skin, and whilst holding the limb in my hand I watched the result of stimulating the ganglion by connecting and disconnecting the battery (using non-polarizing electrodes). While before I could only verify the accomplished change by a dissection, I now saw it arise before me in all its phases. I must first remark that sometimes, but not always, the stimulation of the ganglion is accompanied by a convulsion of the animal, with contraction of the biceps. This appears to me to be partly due to subsidiary currents affecting the neighbouring cervical plexus. Whether the muscle contracts or not is immaterial to the further proceedings; it has, therefore, no relation to the actual changes. These first commence a few moments later, and we perceive that a spot on the surface of the muscle has lost its gloss and appears dull. A moment later fluid appears at this spot, and we begin to see that the surface of the muscle begins to sink under this spot, and becomes rough. This depression extends itself at the inner and outer sides straight across the muscle, while in the middle it reaches up into the fascia, from which it detaches a small narrow point. So, in from three, four, five, to ten minutes there arises a sore, always of the same characteristic form, the bottom of which quickly becomes red, while the walls thicken. Sometimes it stops at this point, but often the sore increases quickly in depth, so that it divides the muscle into two parts. In this case it is always accompanied by considerable hæmorrhage, which fills the wound,

forces itself between the surrounding muscle fibres, and infiltrates the connective tissue. In my former communications, when I had only seen the completed change, I laid undue stress on this bleeding, and on the contraction of the muscular fibres. Now that I have watched the sequence of events these appear to me to bear a secondary character. The primary phenomenon is a softening or liquefaction of the muscular fibres at a definite spot (the localization of which perhaps depends on some detail of muscular growth). After softening, the muscular fibres tear, the result of the natural tension or tone to which they are subjected; and this without any contraction, or stretching, or spasm of the muscle. The leg can be held quite loosely in the hand and watched while this is going on. The fibres tear as a moderately stretched wire tears when it is made red-hot. It is a sudden local change in their elasticity, which deprives them of the firmness necessary to resist even the usual tension. When they are once torn the free ends must spring back, and thus arise the surrounding knots and the defined walls of the sore; the flow of blood into the latter follows as naturally as bleeding in an ordinary wound. The capillaries are too tender to bear stretching across the sore, so they tear too. Here we must take another fact into account: the ganglion is not without influence on the blood-vessels, and these are frequently dilated. This dilatation, although to a certain degree affecting the extent of the hæmorrhage, has nothing of a primary character, as determining what follows. It does not coincide with the muscular changes. The vessels may be dilated in several other places, in the whole skin, in many muscles, even in the same muscles, and, indeed, to such a degree that there may be slight extravasations; but the characteristic sores never occur except in the definite spots in the biceps and psoas. And on the other hand I have seen instances in which there was scarcely any dilatation of vessels, where the sores remained quite pale, and where, even under the microscope, no dilatation nor bleeding could be seen. If the sores are very deep then of course they cannot remain pale, they must bleed.

The microscopic examination shows in the earliest stages, therefore, when the sores just become visible, very peculiar changes in the muscular fibres; depressions form which fill with fluid, whilst the remaining substance lies, as shining, much-contorted bands, in the interior of the sarcolemma. After a time cell-formation and cell-transformation occur, which also, as I am assured by pathological anatomists, bear quite a special character; but which do not appear to me so important, as they also are secondary phenomena.

Let us appreciate clearly what has really been made out. The ganglion is irritated by an electric current which brings about a change in it. Almost immediately there occurs in definite spots, in definite muscles, a kind of dissolving of the tissue which leads to partial liquefaction, and which renders the fibres incapable of resisting the tension to which they are normally subjected. All the rest follows from this. The process is probably by no means so simple, but it agrees with the conception I sketched at the beginning; that the part which has been robbed of its normal power of liberating energy is injured by external forces. At the same time, it indicates that the ganglion cells exert an influence on this power of the muscle by means of which it maintains its metabolic activity in presence of all forces acting on it. It is not improbable that this influence rests upon an interference with the chemical economy of the muscle, for the sudden change in the muscle is more easily understood when regarded as a chemical action. Therefore, definite sympathetic ganglia take part in the chemical economy of definite muscular fibres co-ordinated with them. But how much more remains to be asked? How can we conceive this interference with the chemical economy? As a transference of material, or in some other way? What is occurring in the intermediate links? Does this influence go through the cells of the spinal ganglia and the motor cells of the anterior horns, and does it affect these or spring over them? After reaching the cerebro-spinal system by means of the branches of communication, will it be carried further in its centripetal and centrifugal fibres in the same manner and according to the same laws as are the functional (*i.e.*, the sensory and motor) excitations; and what relation does it bear to these?

These are all unanswered questions, which show that we stand only on the threshold of the problem. We can only advance slowly in so new a matter, because pre-conceived notions and the teaching of the schools leave us in the lurch; but, feeling our way in all directions, we must endeavour to furnish, by our individual researches, foundations on which we may further build up our knowledge. I have performed more than two hundred operations, so as to be able, with some prospect of certainty, to demonstrate the experiment, which I look upon, to a certain extent, as typical of trophic action. In the prosecution of these researches I have too often had to relegate the experiments to the category of the marvellous and the incomprehensible. Anyhow, these actions appear exceptional as long as our minds are prejudiced; but if we take the trouble to follow out the experi-

mental origin of these theories of the schools, and to clear them from misconceptions, we find the contradiction vanish. I have felt compelled to work in this direction myself, and for this reason I have not hesitated to amplify the general remarks in my introduction.

Wertheim Salomonson on Tremor. (*Nederlandsch Tijdschrift voor Geneeskunde*, 1894, No. 1.)

THE author first gives a full account of the graphic methods of studying tremors and a description of the apparatus used in his researches, consisting of a system of two or three Marey's *tambours à répulsion*, which enables him to register every movement or tremor of a limb. He relates eight cases in which he obtained the singular tracings already mentioned in Gowers' "Diseases of the Nervous System," 1888, vol. ii., p. 595, Nos. 6 and 7. These tremors are called by our author allorhythmic or interferential tremors.

Lead poisoning, Parkinson's and Basedow's diseases, hysteria and alcoholism were the diseases of which this tremor was a symptom.

No relation could be discovered between the interferences and the respiration or heart's action; and no explanation of these strange tracings could be found in literature. The author was struck with the resemblance to the tracings (known in acoustics) of membranes swinging under the influence of tones of different pitch; and he supposed the alternating periods in the tracings to be caused by the movements of two different groups of muscles, affected with tremor of different amplitudes. By the following experiment he was enabled to verify his hypothesis. If a muscle is excited by a faradic current the interruptions of which are so slow as not to cause tetanic, but only clonic, contractions of the same duration as the interruptions of the primary current, it is evident that we can produce a regular tremor of this muscle.

By taking two faradic apparatus with slow interruptions of different rate and exciting two antagonistic muscular groups, we re-produce the condition of a member vibrating under the influence of two sets of muscles at different clonus rates. In this way the author experimented with both the sterno-mastoids, and thus obtained oscillations of the head, the tracings of which presented very distinctly and regularly the periods of interference.

That the amplitude of any tremor is determined by the mechanical conditions of the limb affected, can be easily proved by loading the latter with a small weight, whereby the amplitude of the tremor is increased.

Now the amplitudes of the tremors of different limbs in one patient are *almost* equal, and so the interferential periods are not so great as we could expect if the differences in amplitude were greater; and so we can understand that in pathology, if a member is vibrating under the influence of two different muscle groups, the difference in rhythm must be a result of the mechanical conditions of the member.

Another form of tremor, not yet observed or described until now, was seen by the author in a case of Parkinson's disease, and in a case of lead poisoning. He calls it "allorhythmic tremor in space."

In the first case in which the left hand shook continually, he constantly and regularly saw forty or fifty pauses per minute in the movements of flexion and extension of the wrist joint. During these pauses he could observe slight movements of abduction or adduction of the hand in the same joint. These movements were thus really alternating. The pauses were not always so marked as to obliterate every movement of flexion and extension. In this condition the movements were the result of flexion and extension combined with abduction and adduction, and a change in the plane of the movements could be discovered. Now and then these phenomena were less distinct when the forearm shook more strongly. In this tremor, also, could be observed an alternation between movements of flexion and extension and rotation, or movements of abduction and adduction.

The author also registered the tracings of these tremors, and here, too, got allorhythmic tracings in two planes. By combining these tracings and projecting them on a third plane perpendicular to the first two, he got tracings resembling those known in physics as the figures of Lissajous; which are produced by the combination of vibration in two planes, and which can easily be seen in using the kaleidophone of Wheatstone.

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